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ELECTROPHYSIOLOGIC INSIGHT IN OCULAR MOTILITY

GOODWIN M. BREININ, M.D.

NEW YORK, NEW YORK

WITH continuing experience in the application of electrophysiologic techniques to problems of ocular motility, there is accumulating a considerable body of data which has clarified some matters while adding to the complexity of others. An important contribution of these studies consists in their elucidation of objective information in a field which has long suffered from an excess of armchair physiology – although such speculation has often been most perceptive.

The technique of electromyography has been fully described in previous publications.[1-7] It consists in the determination of the electrical discharges of the extraocular muscles by means of fine-gauge concentric electrodes inserted subconjunctivally into the muscle belly. The potentials picked up are amplified and displayed on a multibeam oscilloscope. A permanent record is obtained by motion-picture camera and also by tape recorder. Auxiliary apparatus permits integration of the potentials to reveal the energy discharged in any given interval. This enables us to quantitate and to compare the levels of innervation at several positions and with various movements. It is also possible to differentiate the signals, revealing the composition of complex potentials. In this way, information of considerable physiologic importance has been derived.

It is important to keep in mind that we record the electrical activity of muscle fibers which is discharged by a nerve impulse or action current. The nerve cell, its axon, and the group of muscle fibers that it innervates constitute the motor unit – the functional unit of muscle physiology. This has an anatomical and electrical identity. Disturbances of nerve or muscle tissues are reflected in alterations of the pattern, or configuration, of electrical discharges. Our apparatus thus acts as a wire tap to inform us concerning messages from the brain and the integrity of the neuromuscular apparatus. Electromyography is still a very young technique. This is particularly true in respect to its ocular application. As we gather information, however, our insight into the innervational basis of ocular motility is ever deepening. As a tool for investigation of normal physiology and pathologic variations, it has already become firmly established.

This paper will deal with a number of recent advances illustrating the twofold role of electromyography.

THE ANATOMIC POSITION OF REST

The importance of the anatomic position of the eyes as the basic position upon which converge innervations from many sources has long been recognized.

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Presented at the Joint Meeting of the American Orthoptic Council and the American Association of Orthoptic Technicians, Oct. 14, 1956, Chicago.

In the past, it has been impossible to define this position conclusively, since there was no way to prove the absence of innervational factors. This has now been accomplished by electromyography during general anesthesia.[6] Within a few minutes after onset of anesthesia the electrical discharges of extraocular muscle fall away, reaching complete electrical silence in the surgical plane (fig. 1). At this point, the position of the eyes is entirely dictated by mechanical factors – bony, muscular, fascial. This is the true anatomical position of rest, and it is usually one of slight divergence. This true position is also seen in many patients with marked esotropia, which fact points up the innervational nature of most strabismus. It also demonstrates the active innervational basis for divergence in patients with intermittent exotropia, in many of whom only slight divergence of the visual axes may be noted under anesthesia, whereas a larger deviation is evident during consciousness. If divergence were solely a consequence of diminution of convergence, the eyes should assume maximum divergence under anesthesia; this does not happen.

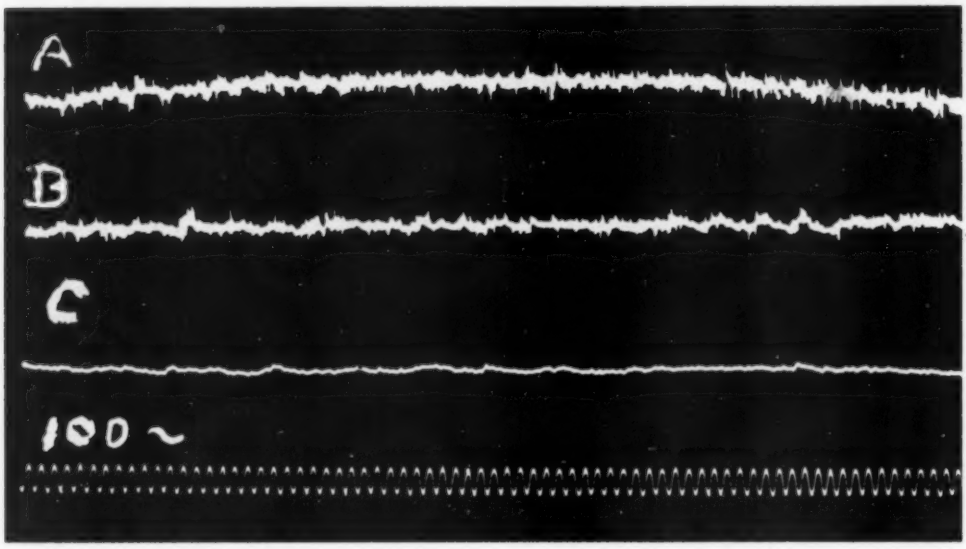

FIG. 1. – Electromyogram of the lateral rectus. General anesthesia. (A) Before anesthesia. (B) Several minutes after injection of pentothal. (C) Surgical plane.

THE INFLUENCE OF DRUGS UPON THE EXTRAOCULAR MUSCLES

The peculiar reactions of the extraocular muscles to pharmacologic agents have been a source of much speculation and experimentation. By means of electromyography, a number of studies have been carried out which dramatically reveal the effect of drugs.

1. Succinylcholine chloride, a drug which paralyzes peripheral skeletal muscle and is widely used in anesthesia as a substitute for curare, actually causes a brief but marked firing and contraction of the extraocular muscles (fig. 2). The resultant increase of intraocular pressure constitutes a strong contraindication to its use in intraocular surgery.[9]

2. Edrophonium chloride (Tensilon®) is an important drug in the diagnosis of myasthenia gravis. The rapid,

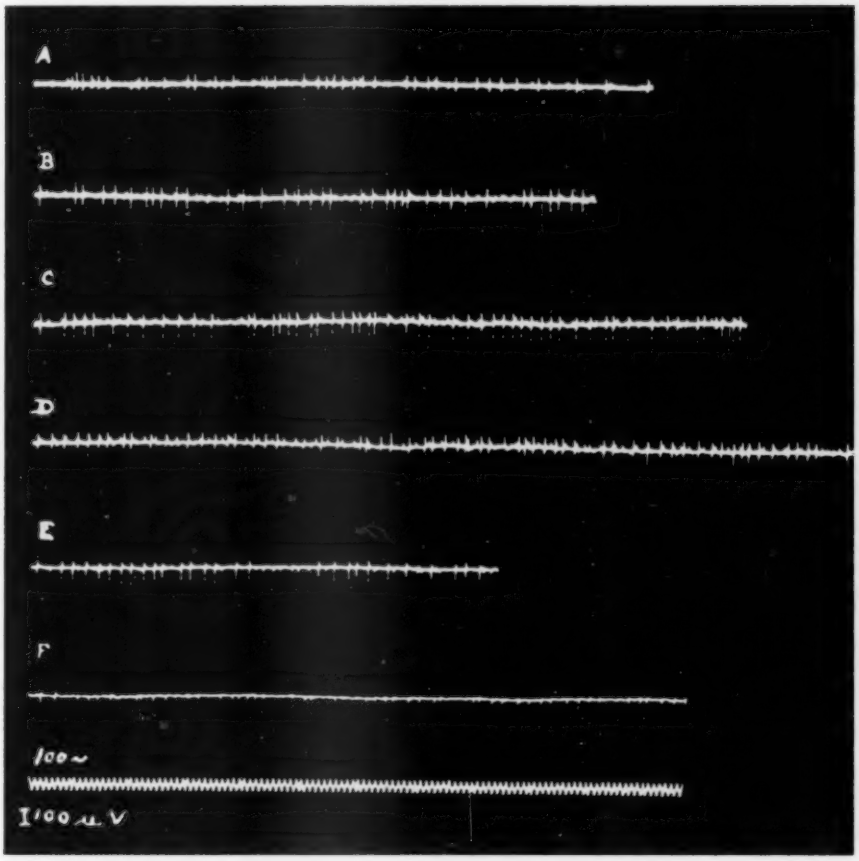

FIG. 2 – Effect of succinylcholine chloride. (A) Before administration of drug. (B, C, D, E) 15 seconds to 2 minutes later; increased frequency and amplitude. (F) After 2 minutes, almost silent.

but evanescent, improvement of the affected muscle that it induces is not always evident by simple observation. When electromyography of the extraocular muscles is used to observe the effect of administration of the drug, there is a brief but most dramatic and well-nigh pathognomonic response, which may constitute the most definitive test for this disease available today.[3] Within seconds the apparently paretic muscle regains a large measure of its electrical activity (fig. 3). This will occur even though gross improvement of motility may be slight or lacking. This use of electromyography may also prove valuable in the pharmacologic management of myasthenia gravis.

THE INNERVATION OF THE EXTRAOCULAR MUSCLES IN VARIOUS FIELDS OF GAZE (KINETICS)

With the aid of the potential integrator, it has been possible for the first time to quantitate innervation in ocular motility. The levels of innervation of the muscles as the eye rotates into various fields of gaze can be precisely compared. This has demonstrated the inadequacy of some long-maintained concepts. From the evidence of innervation, there is little basis for the belief that the vertical recti are active accessory adductors or that the obliques are active accessory abductors in horizontal rotations of the eye. In pure

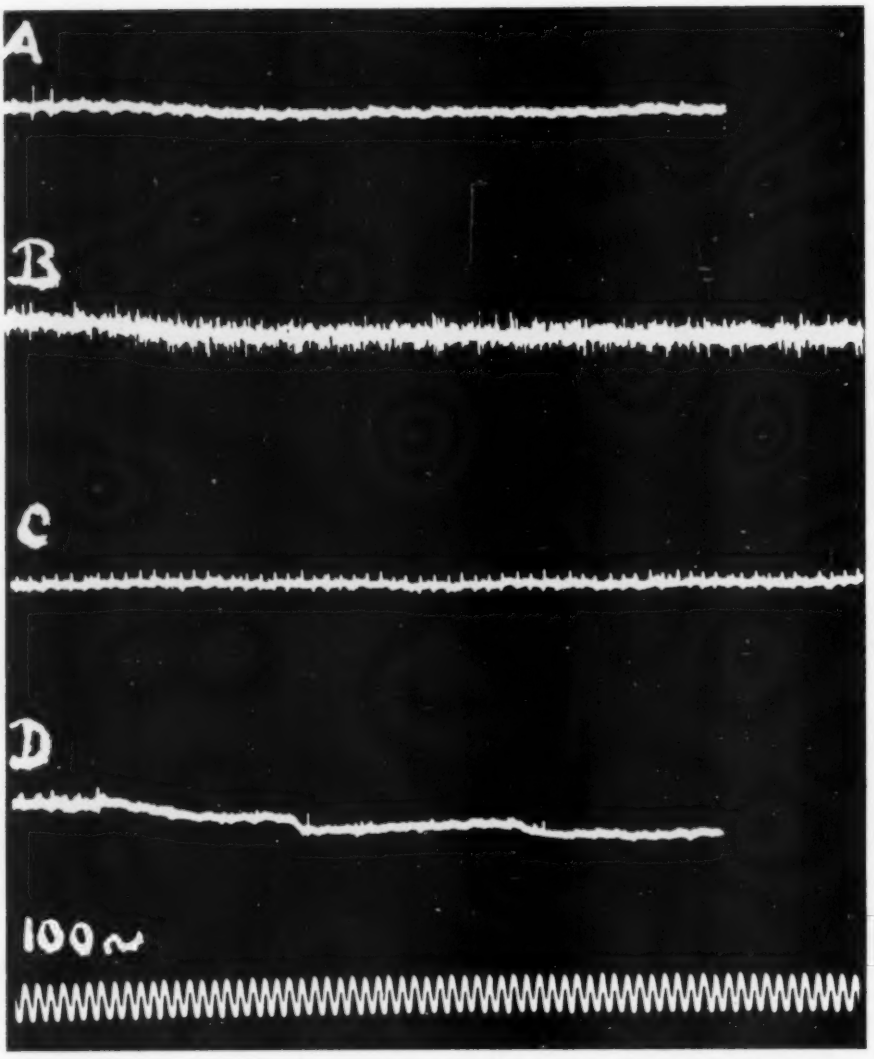

FIG. 3 – Electromyogram of lateral rectus in myasthenia gravis. (A) Almost silent. (B) 30 seconds after administration of Tensilon®; abundant discharge. (C) Single unit discharge one minute later. (D) 2 to 3 minutes later; reverting to baseline. (Amplitude of time signal 100 uV.)

horizontal rotations the tonic activity of the vertical muscles (characterizing the primary position) does not significantly alter (figs. 4 and 5). They maintain a nearly constant level of discharge. Whatever component of abduction or of adduction may be manifested by any one of the vertical obliques and recti will be a consequence of the relation of the muscle plane to the center of rotation of the globe. Thus, as the eye is adducted by the medial rectus the obliques balance each other vertically. The vertical recti, which also balance each other vertically, may develop a component of adduction, due to the change in the relation of their muscle plane to the center of rotation; but it is doubtful that this amounts to very much. When the eye rotates up, the superior rectus and inferior oblique both recruit, reaching a maximum elec-

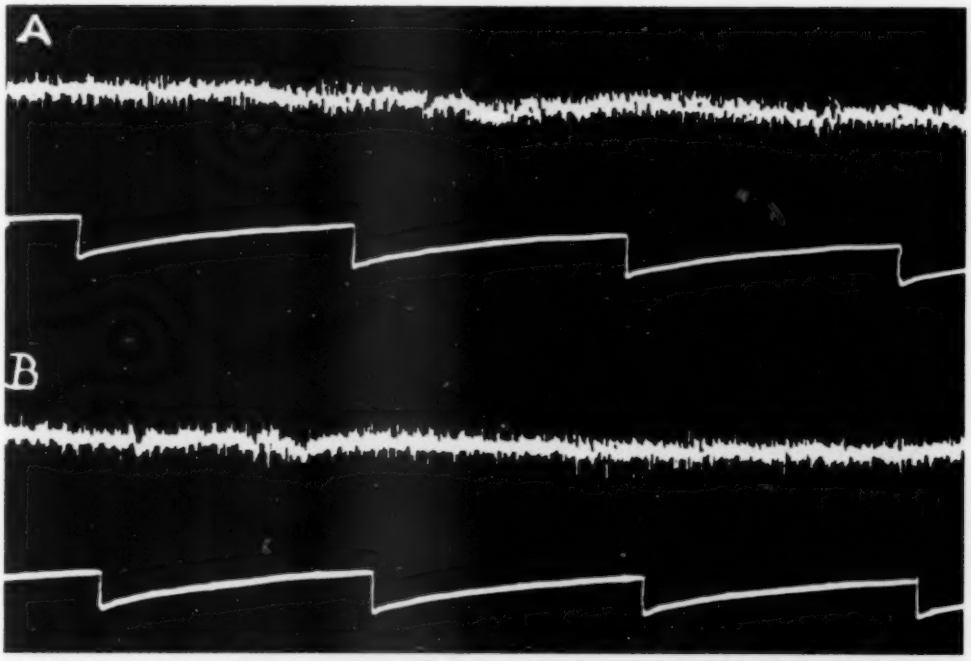

FIG. 4 – Left inferior oblique. No change in horizontal plane. (A) Adduction. (B) Abduction. (EMG above; integration below.)

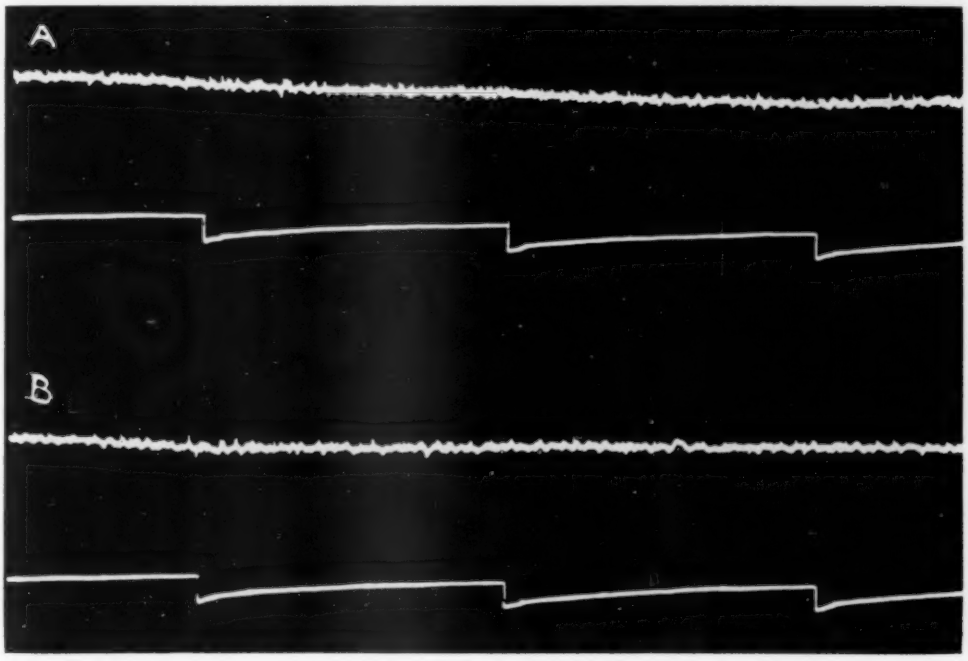

FIG. 5 – Right inferior rectus. No change in horizontal plane. (A) Abduction. (B) Adduction. (EMG above; integration below.)

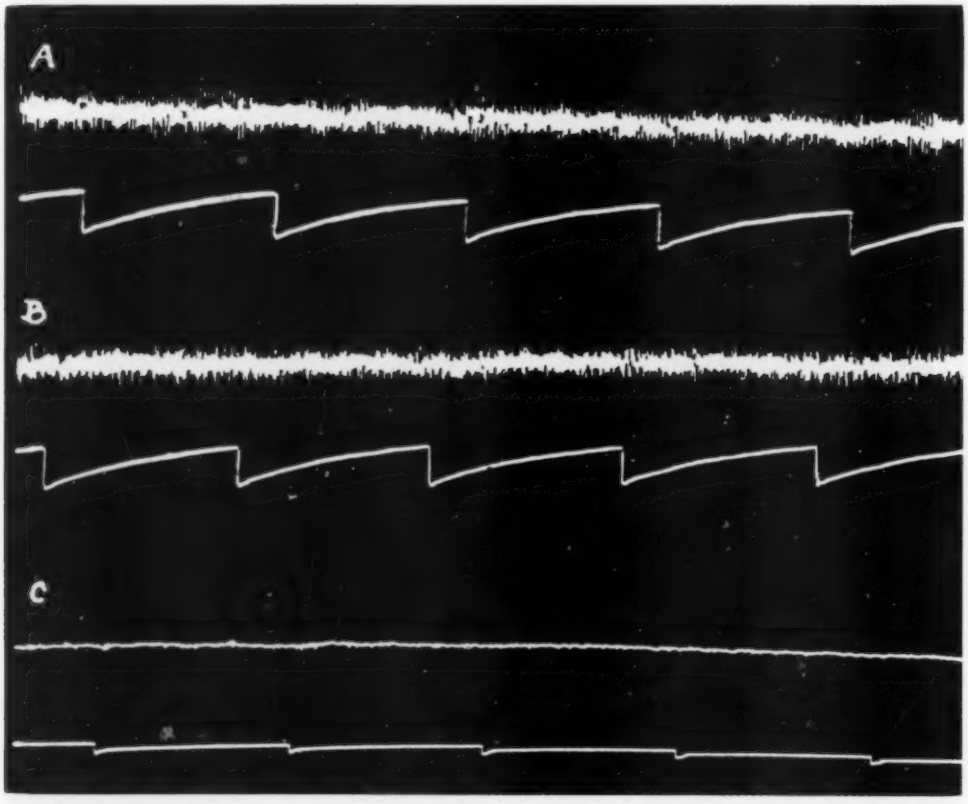

FIG. 6 – Left inferior oblique. (A) Elevation in adduction. (B) Elevation in abduction (slightly less firing). (C) Depression. (EMG above, integration below.)

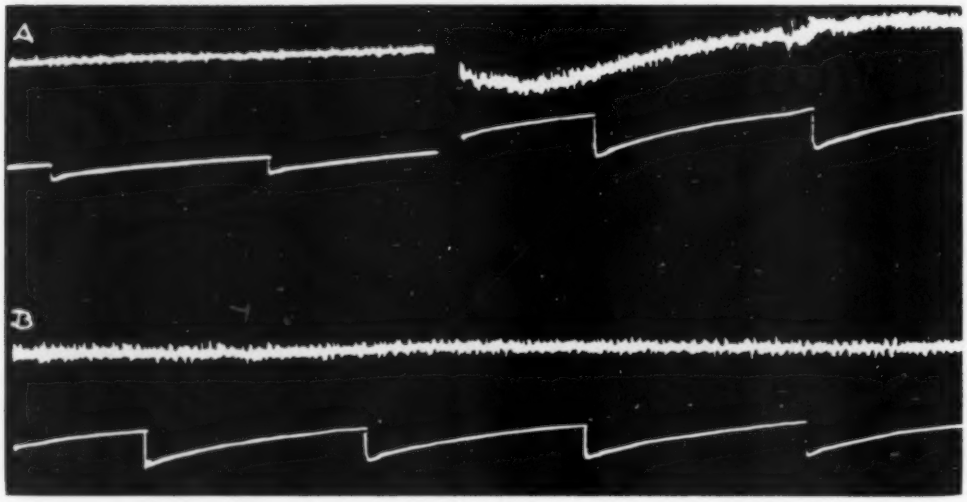

FIG. 7 – Right inferior rectus. (A) Left: primary position; right: gaze down and right. (B) Gaze down and left. (EMG above; integration below.)

trical discharge when the eye attains its maximum excursion. The superior rectus attains its highest level of discharge with elevation in abduction; firing is usually slightly less with elevation in adduction. The inferior oblique attains its highest level of discharge with elevation in adduction; firing is usually slightly less with elevation in abduction (fig. 6). The same features are found when the inferior rectus and the superior oblique are tested in depression (fig. 7). The vertical movers rapidly lose their innervation as the globe crosses the horizontal plane away from their field. Thus, in a tertiary position, e.g., up and out, the lateral rectus contracts while the medial rectus is inhibited; the superior rectus and inferior oblique contract while their opposite numbers are inhibited. There is never any alteration in discharge of the depressors with abduction or adduction as long as the eye is elevated. With sufficient elevation they exhibit no firing at all.

In monocular gaze, straight up or straight down, the horizontal recti also maintain a constant discharge.

The situation in convergence is interesting. It has been maintained that convergence is due solely to the action of the medial recti. The greater adduction of an eye in conjugate gaze, however, has been ascribed to the additional effect of the action of the vertical recti as accessory adductors. Electromyography demonstrates that when the near point of convergence (NPC) has been attained and the gaze is directed to one side, the additional internal rotation of the adducted eye is due solely to an increased firing of its medial rectus (fig. 8). The vertical recti maintain their usual tonic level. This indicates that the role of the vertical recti as accessory adductors is minimal and contributes no more, if at all, to version than to vergence.

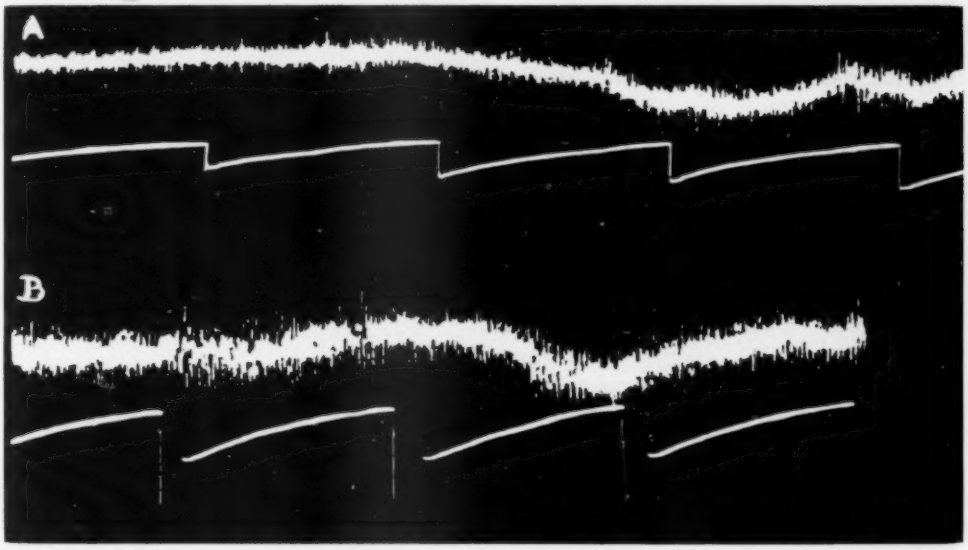

FIG. 8 – Left medial rectus. (A) Convergence to NPC. (B) Gaze right from NPC. (EMG above; integration below.)

The inferior rectus, however, is actively firing during convergence below the horizontal plane. Its level of discharge is related not to convergence but to the degree of depression of the globe. Since intorsion, not extorsion, is said to occur with convergence, the activity of the inferior rectus as an extorter must be overcome, presumably by

the action of the medial rectus as an intorter. This may be a function of the vertical level of convergence.

PARETIC INVOLVEMENTS OF THE EXTRAOCULAR MUSCLES

A fertile field for electromyographic study is that of paretic and incomitant motility disturbances.

1. *Pseudopalsy*

The existence of marked paresis, as distinguished from the presence of anatomical anomalies, can be readily determined. It must be emphasized, however, that minor pareses are at present indistinguishable by this technique.

An example of pseudopalsy is the case of a patient who exhibited a severe esotropia but had practically no power of abduction. It was impossible to obtain a satisfactory history, but it was learned that the strabismus had been present for many years. The electromyogram (EMG) of the lateral rectus was completely normal. The forced duction test revealed limited abduction. The medial rectus was bound to the orbital wall by scar tissue. Because of the normal EMG of the lateral rectus, a surgical procedure involving lysis of adhesions and recession of the medial rectus was done. As a result of the operation, the position of the eye was greatly improved and abduction of about 20 degrees was possible. Electromyography thus provided helpful information on the integrity of the neuromuscular apparatus.

2. *Retraction Syndrome of Duane*

The question of whether the lateral or the medial rectus is responsible for the clinical findings in the retraction syndrome has been widely argued. It seems likely that the same clinical picture may result from affections of either the lateral or the medial rectus. Electromyographic study of several adults has been instructive and permits ready differentiation.[4] The lateral rectus was implicated in both of the following cases:

a. In one case there was almost complete absence of innervation to the lateral rectus (fig. 9). The medial rectus was entirely normal. When the electrode was inserted into the lateral rectus a gritty substance was encountered. Surgical exploration revealed that the muscle was fibrotic and only a few intact muscle fibers were present.

b. In the second case there was a low-level discharge of the lateral rectus in the primary position and no recruitment on attempted abduction. On adduction, however, the lateral rectus increased its firing markedly (fig. 10). This anomalous innervation, manifested when the eye rotates out of the field of the muscle, has been a striking and unique finding in a large number of cases of ocular muscle palsy and is extensively discussed in another publication.[5] We believe that this is a manifestation of muscle stretch revealing a basic proprioceptive mechanism in extraocular muscle. This has nothing to do with position sense, however, and relates only to a feed-back mechanism controlling the level of discharge of the muscle fibers.

3. *Progressive Nuclear Ophthalmoplegia*

The syndrome of gradually developing ocular immobility has been extensively discussed and its causation variously attributed. Several patients with this condition have been studied electromyographically, revealing a striking identity of discharge pattern.[4] The extraocular muscles exhibited a very low amplitude discharge that, on attempted movement into the field of action of the tested muscle, recruited into an abundant but still low-amplitude discharge (fig. 11). This pattern has been described for peripheral skeletal muscle in cases of muscle dystrophy. The histopathologic changes in the extraocular muscle tissue have also been reported as consistent with those in muscle dystrophy. It is therefore felt that the condition is innervationally allied to muscle dystrophy and probably is not a primary neural disease.

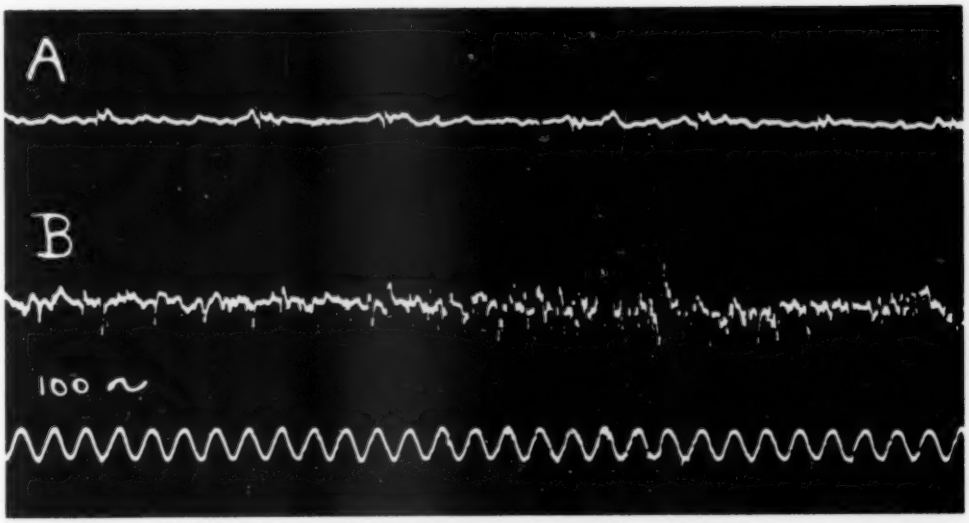

FIG. 9 – Duane's syndrome. (A) Lateral rectus; low grade activity in primary position and attempted abduction. (B) Levator; activity increases on attempted abduction.

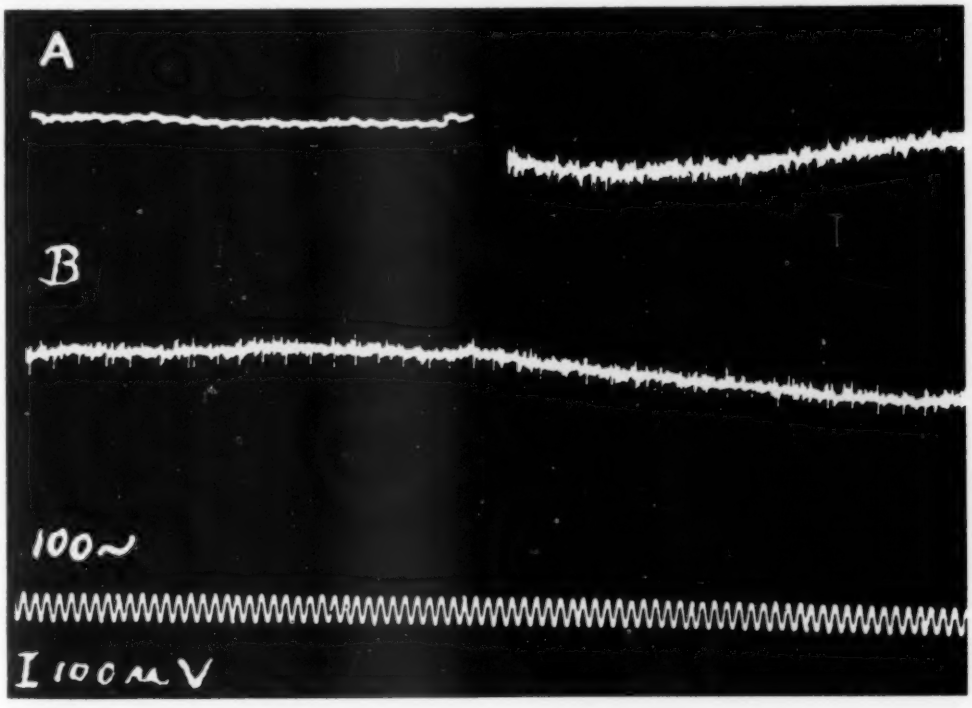

FIG. 10 – Duane's syndrome, lateral rectus. (A) Left: no activity in primary position or on attempted abduction. Right: anomalous recruitment on adduction. (B) Anomalous recruitment.

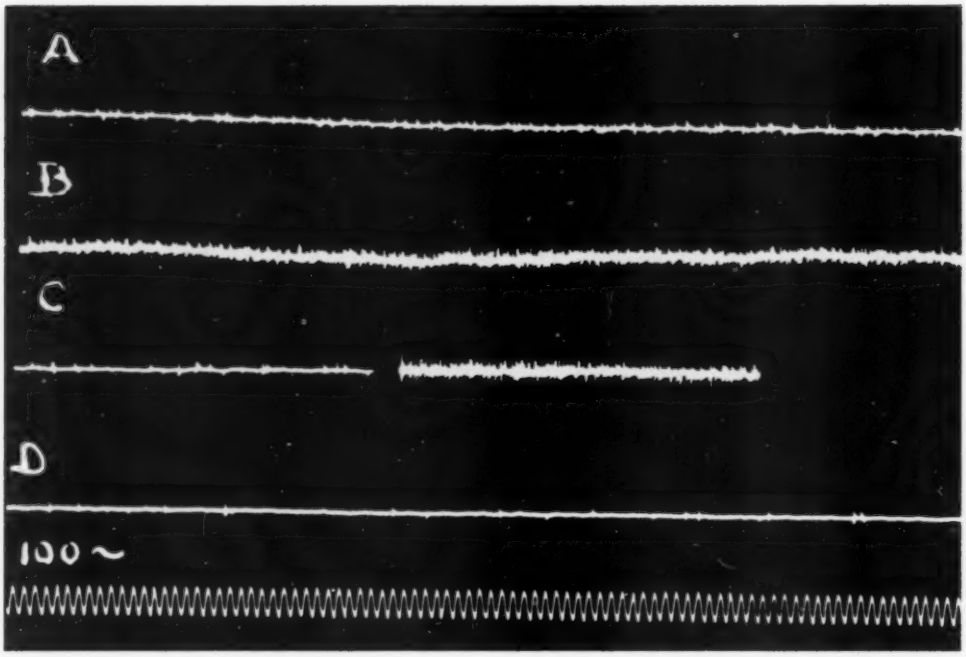

FIG. 11 – Electromyogram of lateral rectus in progressive nuclear ophthalmoplegia. (A) Primary position. (B) Abduction: abundant recruitment, but low amplitude. (C) Left: gaze ahead; right: attempted movement into the field of action of the tested muscle, side of lateral rectus. (D) Primary position; low-grade activity. (Amplitude of time signal, 100 uV.)

4. *Neurogenic Lesions*

The presence of denervation, indicating disease of the lower motor neuron, is revealed by fibrillation potentials (fig. 12). If reinnervation occurs the number of fibrillations lessens and ultimately may cease, concomitantly with the appearance of normal motor units. Polyphasic units indicative of disturbances in the motor unit may persist indefinitely or also gradually disappear (fig. 13). An extensive discussion of these problems has recently been published.[4]

5. *Aberrant Regeneration of Third Nerve Fibers*

Following oculomotor nerve palsy, a characteristic syndrome may develop. On any attempted movement except abduction, the upper lid retracts, the eye turns in, and the pupil constricts. This is attributed to aberrant regeneration of the third nerve fibers which grow into the various branches of the nerve indiscriminately.[4] Thus, any attempted movement other than abduction is neutralized, except for lid retraction and adduction – since these are the only unopposed functions in the third nerve complex. Electromyographic analysis has revealed simultaneous discharge of all the muscles of the third nerve complex (fig. 14). This completely explains the clinical picture on an innervational basis.

NYSTAGMUS

A patient with a brain tumor was found to have a loss of voluntary vertical gaze, associated with retraction and convergence nystagmus. Electromyograms of the medial and lateral recti revealed almost simultaneous dis-

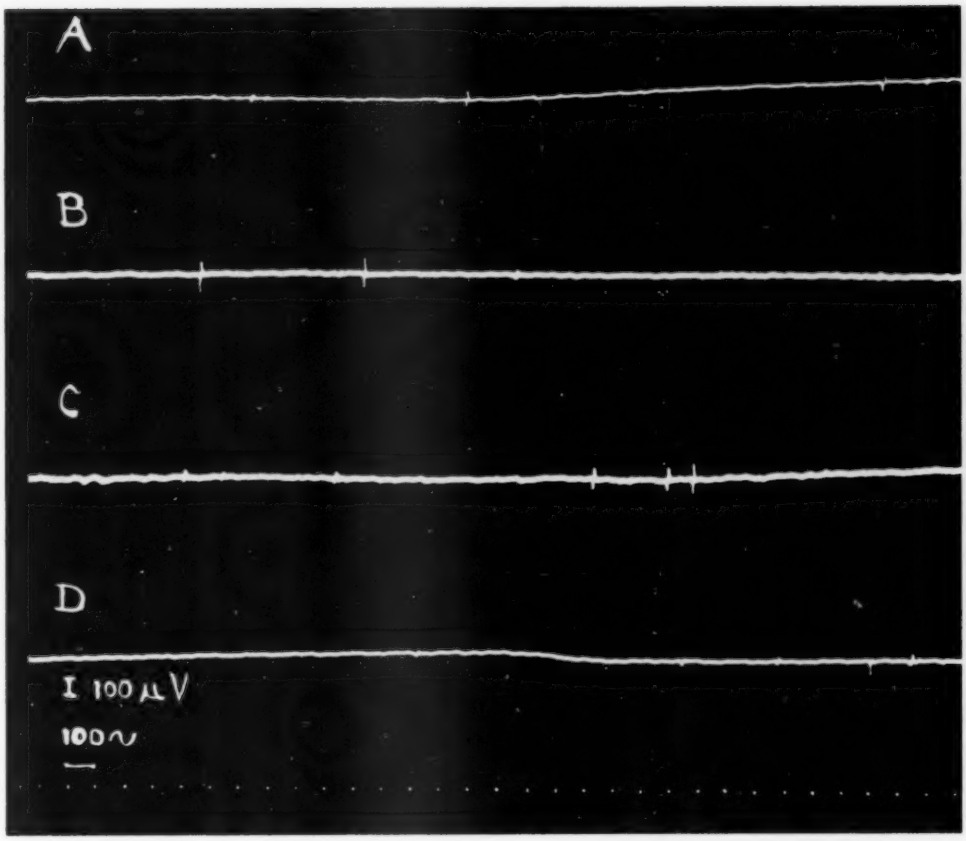

FIG. 12 – Complete third nerve palsy. (A, B) Levator fibrillation. (C, D) Medial rectus fibrillation.

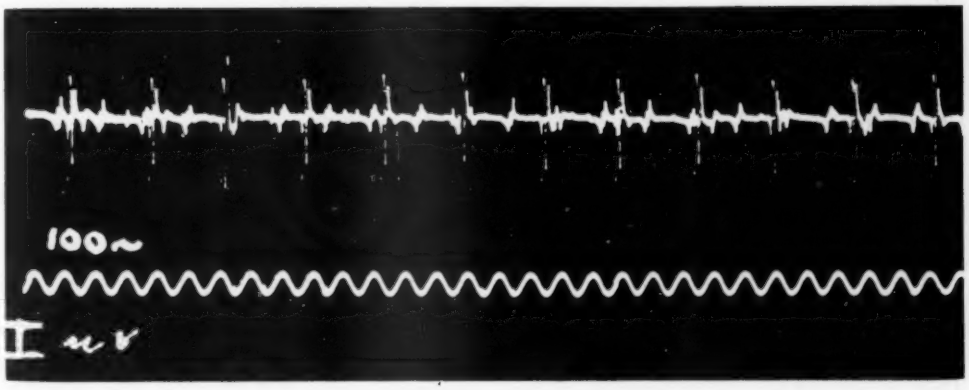

FIG. 13 – Polyphasic potentials in abducens palsy, lateral rectus.

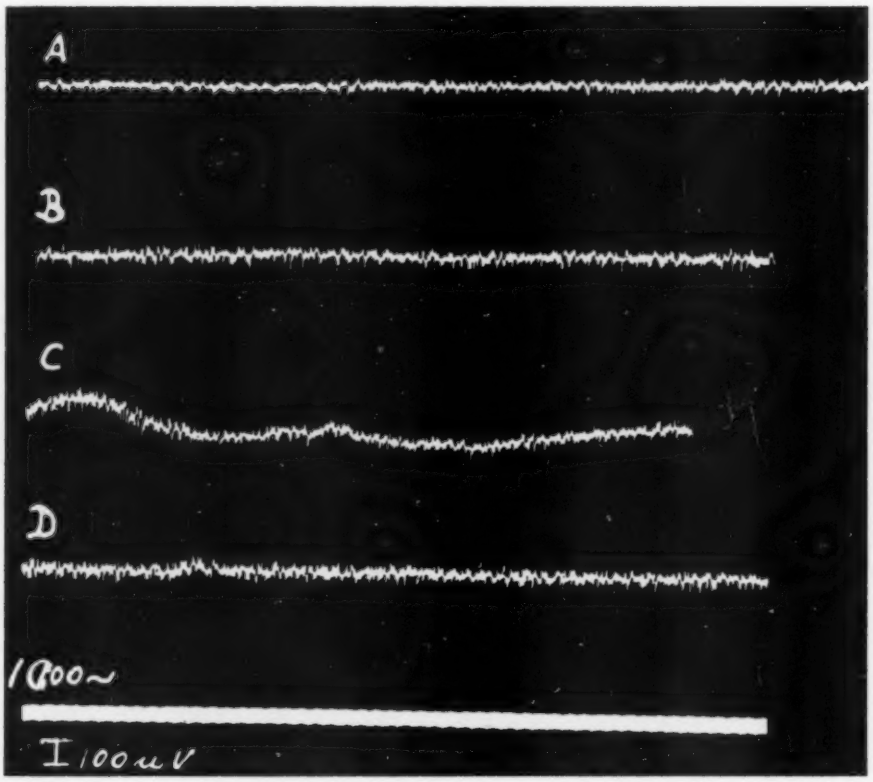

FIG. 14 – Aberrant third nerve regeneration, inferior rectus. (A) Primary position: low amplitude discharge. (B) Gaze down: increased discharge. (C) Gaze in: even larger discharge. (D) Gaze up: discharge increased over initial level.

charge of the antagonists, with resultant retraction of the globe (fig. 15). In most nystagmus the contraction of the agonist is accompanied by inhibition of the antagonist. This relationship was lacking during retraction nystagmus and was faulty during the convergence nystagmus. A central disturbance of the reciprocity mechanism is thus evident.

The electro-oculogram (EOG) is a record of the standing potential of the eye and exhibits variations during movement. Simultaneous electromyograms and electro-oculograms permit a detailed analysis of the innervation to the eye and of the resultant movement. This is advantageous in studies of nystagmus because the patterns of the two tracings give a more nearly complete picture of the underlying disturbance (fig. 16).

STUDIES OF VERGENCE

The use of the integrator enables a truly quantitative approach to problems of innervation. This is proving advantageous in the study of effects of prisms and lenses. The influence of convex and concave lenses on accommodative vergence is made evident in a simple and quantitative fashion. Increasing the stimulus to accommodation by the use of increasingly powerful minus lenses is reflected in higher levels of innervation to the medial rectus of the occluded eye (fig. 17). The fixing eye, of course, shows no alteration in accordance with the demands of fixation. This

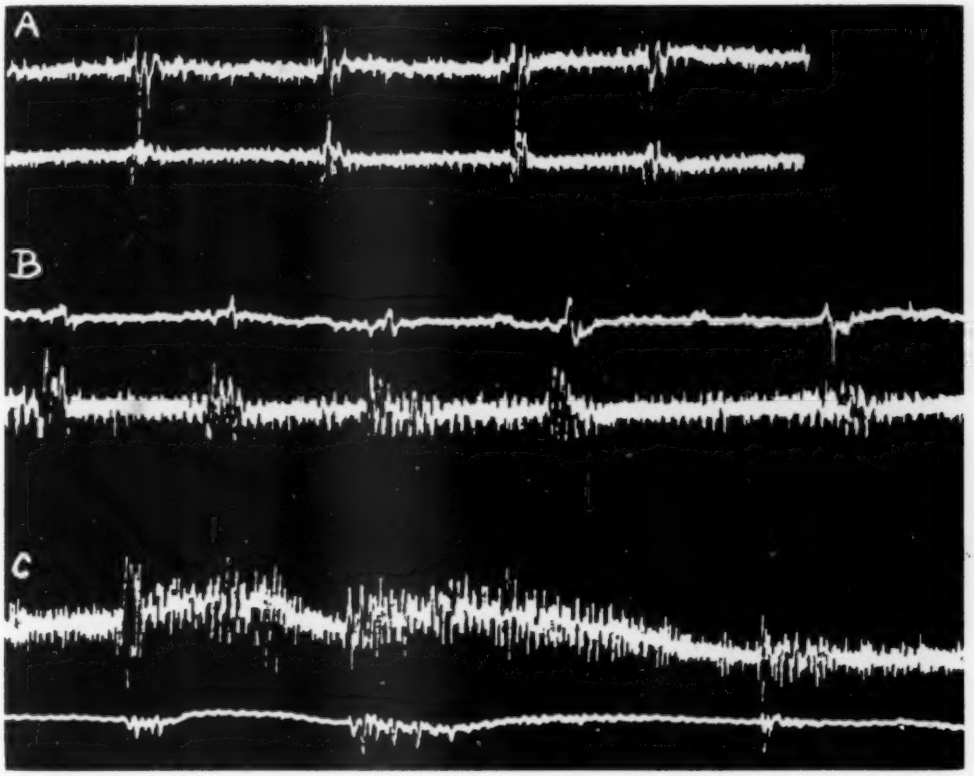

FIG. 15 – Retractory and convergence nystagmus, right eye. Simultaneous recruitment: lateral rectus above; medial rectus below. (A) Primary position: retractory nystagmus. (B) Convergence nystagmus. (C) Abduction.

has been described previously.[2] It is anticipated that this technique will prove productive in analysis of problems of accommodation and convergence.

PROPRIOCEPTION IN EXTRAOCULAR MUSCLE

The existence of a mechanism whereby stretch of the ocular muscle fiber influences the tonic level of innervation to the muscle has been demonstrated.[5] The afferent mechanism of spindle discharge in extraocular muscle has been conclusively demonstrated by Cooper, Daniel, and Whitteridge.[8] We believe that the motor mechanism is evident from our studies. This evidence may be summarized as follows: the characteristic tonic level of firing present in the primary position disappears when the muscle is cut free from the globe, its fascial attachments are severed, and it is allowed to freely retract. The delicately graded reciprocal innervation of antagonists is altered when the muscles are cut free (as after enucleation). This may be likened to an ataxia. There are no phasic or tendon-jerk reflexes, but a static mechanism maintaining a level of discharge appears to exist.

In addition, the phenomenon of anomalous innervation in many paretic muscles, previously mentioned, is considered to result from a stretch mechanism made manifest through the paresis. Passive rotations away from the field also elicit an anomalous discharge in most, but not all, cases.

Although proprioception in extraocu-

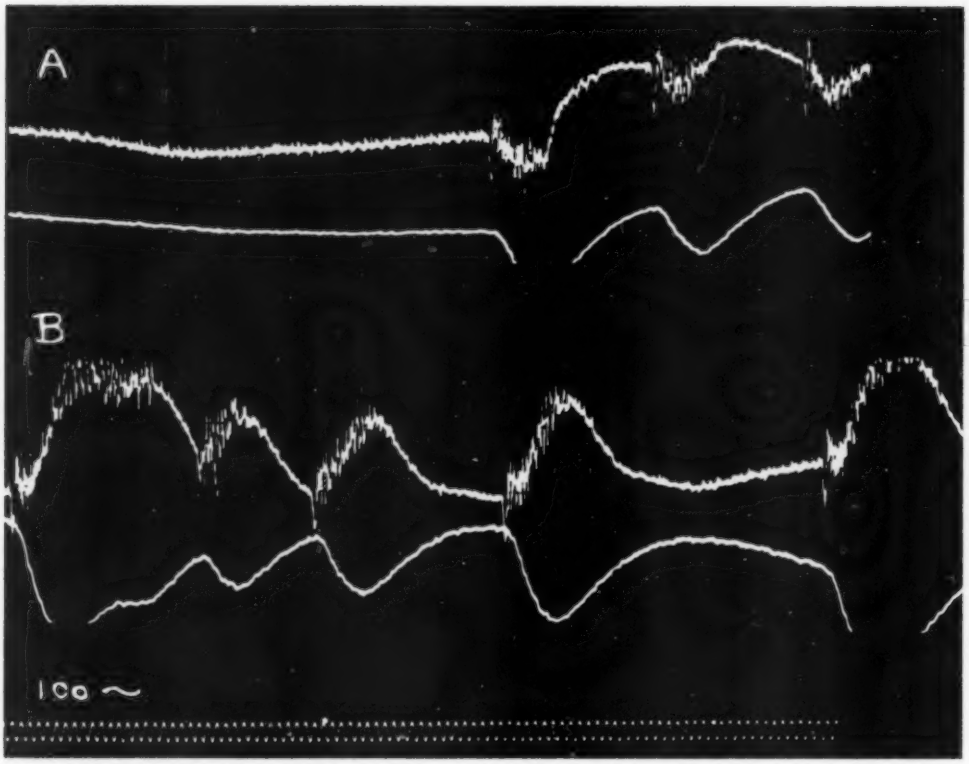

FIG. 16 – Nystagmus; simultaneous electromyogram and electro-oculogram. (A, B) Upper tracing, EMG of medial rectus; lower tracing, EOG. Relationship of innervation and consequent movement clearly indicated. (Amplitude of time signal, 100 uV.)

lar muscle differs from that in peripheral skeletal muscle, it is nevertheless present and presumably helps maintain the delicately balanced level of innervation. It is thus evident that extraocular muscle retains a fundamental kinship with the other skeletal muscle.

SUMMARY

These have been some of the insights into ocular motility which have been gained in our laboratory during the past year.

The application of new research techniques to the problems of ocular physiology is a fascinating and rewarding experience. While our approach has been directed toward elucidating basic physiologic processes, there have been numerous by-products of considerable practical value. Thus far we have scaled only the foothills in ocular motility. From this vantage point we see no lands' end or "broad Pacific," but only range upon range of new and unsuspected problems, ever mysterious, ever challenging. There is no Mount Everest in the quest of science. For this we may well be thankful.

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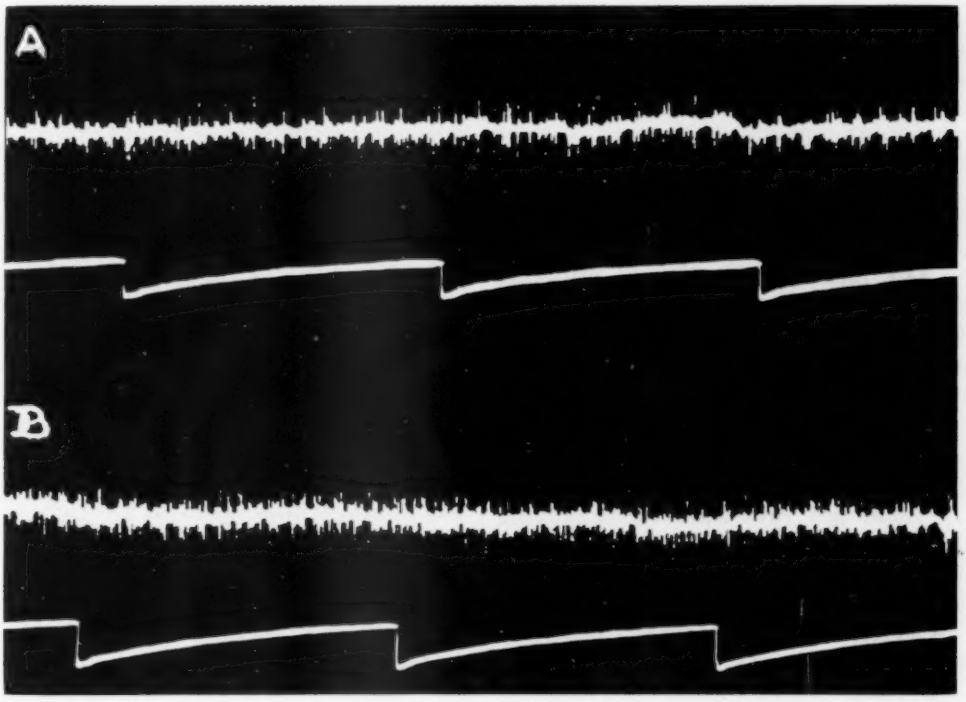

FIG. 17 – Accommodative vergence. Medial rectus, left eye. (A) Primary position. (B) with –1 diopter lens before the right eye. Note increased firing and height of integrator discharge. (EMG above, integration below.)

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A NEW APPROACH TO CLASSIFICATION OF ORTHOPTIC CASES

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THE ideal classification of orthoptic cases is in units of one. Each patient offers individual problems in treatment. However, for convenience in discussing orthoptic problems and treatment, it is helpful to group certain types of problems, and devise techniques for solving them.

There are two ways of classifying orthoptic cases which are widely used. One is on the basis of diagnosis: a patient has a phoria or a tropia; he has an external or an internal squint; his disability is paretic or innervational in type, and specific muscles are affected. To make such classification requires an understanding of ophthalmology. The technician must either have extraordinary competence in ophthalmology or rely on the ophthalmologist to make the classification for her. This method of classifying cases is not an orthoptic approach.

A second method of classification is by Worth's three grades of fusion: simultaneous perception; fusion with amplitude; and stereopsis. This is an orthoptic approach and can be used by technicians, but modern progress in orthoptics has proved that it leads to confusion in application. It groups together a variety of cases which need to be differentiated on the basis of required treatment, and if this classification is relied upon, it leads to treatment which is inaccurate and not directed specifically enough to the individual problem. Attempts are made to stretch the words to fit the case or to give special meanings to the words; a common example is the phrase "superimposition without fusion." The use of this phrase for classification does not explain in which way simultaneous perception occurs in the patient under consideration. It may occur in a variety of ways, depending upon the location and character of the images, and how the brain interprets them.

One type of target used in the amblyoscope (and stereoscope) is called the simultaneous-perception target. It consists of dissimilar pictures which must be fitted together to make a complete or perfect picture. Well-known examples are the lion and the cage, the baby and the crib, the flowerpot and the window. In the absence of simultaneous perception the targets never fit, nor do they have any spatial relationship. In the presence of simultaneous perception, if the images are on corresponding areas they will fit, either normally or anomalously. In the presence of suppression no second image is perceived; there is no second image to relate. In the presence of simultaneous perception of images on noncorresponding areas, the two targets do not fit but do have a specific location in reference to each other.

The instinct for coordinating the images from the two eyes is very deep. In the absence of simultaneous images the patient often tries to perceive and describe a relationship which is simply nonexistent. Such descriptions should

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not be considered as reliable observations of facts, as they are frequently only hoped for or imagined perceptions. Children are sometimes completely frustrated by their inability to describe spatial relationships, especially when they do not exist at all or are so fleeting that careful, leisurely observation is impossible.

The use of simultaneous-perception targets permits a study of the fixation skill of the patient, of his correspondence, and of his ability to describe spatial relationship. Since the material is, by definition, nonfusible, no evidence about fusion can be collected, although much evidence about the patient's capacity to fuse can be deduced.

The second kind of target is the fusion target. It presents fusible material to the patient. Fusion targets may be presented to the patient in two ways: on corresponding areas and on noncorresponding areas. If the targets are presented on corresponding areas the patient may suppress one image (in whole or in part, constantly or intermittently). When suppressing, the patient does not have simultaneous perception and he cannot fuse. If he does not suppress, then the images are fused, according to the definition of fusion: fusible images received on corresponding areas. There is no need of the word *superimposed.*

Fusion, however, is no guarantee of amplitude. A patient with simultaneous perception on corresponding areas of fusible material has fusion, but he may not have Worth's second grade of fusion, which is fusion with amplitude. It is the character and location of the two images, not the visual function, which differentiates simultaneous perception and fusion, since simultaneous perception can exist when the images fall on noncorresponding areas. If simultaneous-perception targets are used, the perception is of "not fitting"; if fusion targets are used, the perception is diplopia. The kind of disparity evidenced by use of the simultaneous-perception target does not exist on casual seeing. It is highly artificial, and since it is new and different to the patient, it gives the technician an opportunity to teach the patient correct observation under highly controlled situations on the amblyoscope; it dramatizes spatial relationship and makes correct observations easier, with fewer preconceived ideas to break down. The perception of diplopia is a normal function, but it is often conditioned by the patient's previous experience and vocabulary, resulting in incorrect conclusions about spatial relationships. Results with material using simultaneous perception are easier for the technician to evaluate, but material requiring actual fusion is easier for the patient to use.

The technician is constantly trying to discover, and so classify, two aspects of a patient's binocular functioning — the motor and the sensory. If the patient has *simultaneous perception,* he has sufficient motor control to fix with one eye and maintain the other eye in stable relationship (angle) so that two images are received; his sensory apparatus perceives both images and gives them relationship — he does not suppress and is not too inexperienced (immature) to relate them. But the phrase "has simultaneous perception" gives no indication of the conditions under which this status exists. It explains nothing about whether the images are perceived only when on corresponding areas, or only when on noncorresponding areas, or under what conditions of stimulation, or with what size or kind of target. In order to classify cases usefully, the conditions under which the patient has simultaneous perception should be fully explained; conversely, if a patient does not have it, the conditions under which it was attempted should be explained. Failure of the patient to have simultaneous perception under certain conditions is not proof that he would

fail to have it under all conditions. In order to classify a case for efficient treatment of the patient the motor situation under which he is tested should be described. The basic motor skill is fixation. I have seen patients who had been considered as not fusing but who were unable to fix; consequently no evidence about their fusion status could have been obtained. When these patients learned to fix, they did fuse when fusible images were presented to corresponding areas under sufficient stimulation to prevent suppression. Such patients badly need orthoptic training for skill in fixation. They are not ready for exposure to fusion, much less to fusion with amplitude. They do not perceive fusion because they lack the requisite motor skill of fixation, and no evidence about their sensory status can be collected. The ability to hold two images in stable relationship is the first step in fusion skill. The images may be like or unlike; they may be on normal or on anomalous corresponding areas; or they may be on noncorresponding areas. In such cases the perception on casual seeing is diplopia; on the amblyoscope the perception is unfitted SP (simultaneous-perception) targets or diplopia of fusion targets.

Many patients get simultaneous perception when the images are on noncorresponding areas and suppress one eye when the images approach or reach corresponding areas. This is quite easy to detect on the amblyoscope if the patient is a monocular squinter with a preferred eye. If the patient alternates, more skill is needed to detect whether there is a loss of motor stability when he is suppressing, i.e., whether there is a motor change of the fixing eye or a sensory change only, from perception to suppression. If a motor change occurs, the skill of the technician is needed to improve motor control. If the error is sensory, a different technique is required. The technician should know which kind or class of error she needs to treat.

The next step in fusion skill is vergence control. This is Worth's second grade of fusion: *fusion with amplitude*, as demonstrated on the amblyoscope or by the ability to maintain fusion for different distances fixated on casual seeing. The lack of vergence control does not mean the absence of fusion. It is not lack of vergence control which prevents fusion, but three other conditions: (1) lack of fusible material, as when SP targets are used; (2) unstable fixation, which prevents the reception of two images for sensory interpretation; and (3) suppression.

There are two kinds of vergence movements which should be differentiated in analysis and study: (1) true vergences, associated with fusion, and (2) reflex vergence movements, resulting from fixation with one eye while the other is suppressed (or covered). A change in the posture of the eyes, such as takes place in many cases when the patient changes the distance fixated, may or may not be associated with fusion. In claiming that a patient has amplitude the technician must offer evidence that fusion exists, or else qualify her diagnosis with "close to normal vergences" or some other suitable phrase; indicating that fusion evidence was not collected. Vergence control should be analyzed for both speed and endurance: how promptly can the patient change his fusion angle, and with what endurance can he hold it?

Stereopsis is Worth's third grade of fusion. Stereopsis is innate, but accurate perception of it requires experience and adequate vocabulary for evidence. The presence of stereopsis proves that the patient's fixation on the two images is sufficiently stable for interpretation and that his experience and vocabulary are adequate for reporting the situation to the technician. Lack of stereopsis may be due to either un-

stable fixation or immature observation. It does not necessarily mean sensory disability. The experience of stereopsis by the patient when using the amblyoscope is not proof of fusion for casual seeing; it does not mean that he has achieved the ultimate in binocular skill and is ready for discharge. Patients may have stereopsis under the highly controlled situation on the amblyoscope and not have vergence control for casual seeing.

My approach to the classification of cases is to find out, first, whether the patient can receive one image with sufficient stability to interpret it, such as by identification or color. Second, can he receive two images, and if so, under what conditions of location, size and stimulation? Third, are his sensory interpretations of such images normal or anomalous; has he accurate spatial perceptions of the relationship of the two images, or is he conditioned to erroneous perceptions (not necessarily anomalous)? Fourth, is his fixation stable enough for stereopsis? Fifth, has he vergence control; is it associated with fusion or reflex only; is it swift and stable?

The final evaluation of the patient depends upon whether he can apply his binocular skill in casual seeing or only under controlled conditions and motivation.

At each step in the development of binocular skill it is important to determine the patient's status, to evaluate his need for further progress, sensory or motor, and to balance his need against the difficulty he will have in achieving binocularity.

1. Has he sufficiently stable fixation for fusion; for stereopsis?
2. Does he accept fusion without suppressing? Is it normal or anomalous?
3. Can he expect enough vergence control for his needs in casual seeing?
4. Has he the speed and endurance necessary for casual seeing?

Orthoptic lessons should be clearly directed at each skill. Sometimes two skills can be developed simultaneously; this is especially true of the motor and sensory aspects of each fusion step.

The efficient treatment of an orthoptic patient is dependent on an evaluation of his binocular skill, including both sensory and motor aspects. We must remain open minded about orthoptic progress and retain a flexible attitude toward the problems of treating each patient. He should not be forced into a diagnostic mold; instead he should have individually planned opportunities for progress adapted to the level of binocular skill manifested at each visit.

BROADER HORIZONS IN ORTHOPTICS

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IN medicine we have seen the variation from favor to disfavor of many advocated therapeutic mechanisms; often the cycle has repeated itself time and time again. Orthoptic training is no exception. The groundwork of what we know today as orthoptics was laid by du Bois-Reymond in Mueller's archives in 1852, and by Mackenzie in his treatise on diseases of the eyes in 1856. It was during the latter part of that century that Javal wrote on the subject, bringing to it explanations based on sound scientific principles. In his manual on strabismus, published in 1896, Javal was somewhat less enthusiastic than in his earlier years. He is reported to have stated that he agreed with von Graefe, in Javal's earlier years a most enthusiastic supporter of orthoptics, who remarked that people were not worth so much trouble. Be that as it may, Priestly Smith in 1896 took up the training of squint cases with his "fusion tubes." And around the turn of the century there was quite a flurry of activity; in England, by Worth and Maddox; in France, by Remy; and, in the United States, by de Schweinitz.

Then came another lull, so that the next period of enthusiasm can pretty much be dated as beginning in the 1930's and coming on up to the present. That the field has grown considerably in recent years cannot be questioned. The reasons for this growth and present wave of popularity are perhaps not so easily ascertained.

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It must, of course, be remembered that much of the growth of the whole field of visual physiology has been in the hands of persons without basic orientation to the medical profession, per se. I am thinking particularly of men primarily trained in physics, or in physiology. Certainly, the phenomenon of sight should arouse the curiosity and interest of an inquiring mind. Much of the work in this field has come from the basic-science laboratories of colleges and universities. However, the influence of practicing optometrists, as a licensed group, has to some extent followed the growth in popularity of "eye exercises." One cannot help but wonder if they have not been responsible for much of the "selling" of the concept of eye exercises, particularly to patients who are opposed to surgical intervention as a means of solving their squint problem. Academicians and physicians, alike, have been impressed with the work of the Dartmouth Eye Institute, which, after eighteen years of intensive research, terminated its study in 1947. The following physicians were at one time or another members of the Institute: Alfred Bielschowsky, Hermann M. Burian, Elmer H. Carleton, Werner Hersau, Walter B. Lancaster, Arthur F. Linksz, Paul W. Miles, J. Miles O'Brien, and Kenneth L. Roper. One can argue about the "chicken and egg" relationship here; did the interest of these individuals in this aspect of visual problems come first, and then the Institute, or vice versa? In any event, we must all agree that the existence of

this organization certainly furnished a spark plug in the field.

Over the past fifty years the medical specialty of ophthalmology has undergone considerable evolutionary change. In the old days, as you may know, specialization was primarily by exclusion. That is, after a physician had established his practice, he began to specialize in certain fields, then he simply dropped other phases of his practice; so, in a few years, after limiting his practice to one thing, he became, ipso facto, a specialist. A little later came the addition of the "short course" to this method of specialization. Only during the past twenty-five to thirty years has come the concept of specializing by additional formal training and practice rather than by exclusion. In ophthalmology, specialization by exclusion, as you would quickly reason, bred a type of practitioner primarily interested in the medical and surgical aspects of ophthalmology, since these were more closely akin to that practice which he had previously known. In the "short course" era, the short course was usually concerned with refraction under cycloplegia by the "sees best" method—this, of course, to the consternation of the young hyperope. The point is, many physician eye specialists practicing in this period had too little opportunity to become acquainted with the basic studies in visual physiology and fusional mechanisms, hence, they did not become very interested in these facets of ophthalmology. Certainly, in the past twenty years the minimal acceptable standard for physicians working in the field of eye care has been considerably raised, and with it, a much larger number of ophthalmologists are interested in, and dedicated to the work in which you are our partners.

I should like, now, to approach the subject from a different point of view, and discuss briefly some other changes which have occurred over the past half century.

We have changed from a rural to an urban population; with this has come the great increase in the percentage of persons in the skilled and semiskilled trades. Between 1910 and 1950 the percentage of the male work force in the skilled and semiskilled trades has increased from 28 to 79. For the higher skills, increased visual demands, especially demands on stereoscopic vision, are made. Furthermore, we have now seen the passage of workman compensation laws in every state of the Union. This, of course, is simply one of the many fields in which certain aspects of responsibility to the employee have been assumed by the employer. As you know, many types of eye difficulty, including industrial injury, do serious things to the binocular mechanism. Thus, we now find many employers screening prospective employees; and we see the mass utilization of such gadgets as the Ortho-Rater, designed to test and evaluate visual acuity economically and by persons with minimal training in the use of the machine. More and more we are having to give a quantitative determination regarding visual losses and deficiencies that can be understood in courts of law before nonmedical persons.

Another point worth mentioning has been the increase in public contact with stereoscopic observational mechanisms. 3-D is certainly a household word today. One has only to stand around a camera-store counter a few minutes to note the amount of public interest in cameras that will take three-dimensional pictures. The motion pictures have had a recent flare of activity along this line, but present trends seem away from true three-dimensional methods in favor of the "big screen" approach.

Perhaps of more interest from our point of view is the now widespread use of stereoscopic devices in industry. In this town we have a large number of men employed by a company which manufactures maps, using consecutive photographs made from an airplane. These men must focus two pictures at a time, using a red-blue projector-and-glasses system. Training men to run the machines used in this work is expensive, for the value of a man as an operator cannot be clearly ascertained for at least six months. Thus, the cost to industry simply to find out if a man can do this type of work is considerable. To employ a man for several months, simply to discover that he is unable to focus his projections sharply in making the contour map and is thus inadequate for the job, proves quite costly to the company in the long run. This is the situation that arises after sectional maps of a large area have been drawn; then the adjacent finished maps are sent to a room for "bridging." This simply means hooking the sides of the maps together. But what if they don't coincide? Aside from the observation that "somebody goofed," we must also remember the fact that someone else paid for a lot of salaries, equipment use, etc., on work that must be completely redone. In spite of the many rather serious economic ramifications, the pre-employment screening for this work is practically nil. In addition, the machines that these men use are far more critical than the usual diagnostic instruments. Our local orthoptist, Miss Mimms, and I worked out a tentative controlled series for this company, but little has been done, since it has for some months not had occasion to train any new operators. It is a fascinating problem and, in my opinion, deserves a considerable amount of study in an effort to develop a simple method of picking accurate plotters.

We have seen the rise of a population which travels not on horseback and by foot but, to a large extent, in vehicles under the control of individual citizens – primarily in the motor car and, to some extent, in the airplane. The number of automobiles in 1900 was about 4,192. Today the number is about 5.5 million. Today we have over 72 million car and truck drivers in this country. Likewise, the number of licensed aircraft pilots has risen from 33,706 in 1939 to about 596,128 today. With these changes have, of course, come legal restrictions that are devised to protect us from ourselves; so that to drive an auto in most states you must have a license, and to have a license in many states you must be able to read an eye chart. Likewise, the present eye examination prescribed by the Civil Aeronautics Administration and the Civil Aeronautics Board has no less than twelve separate types of tests, each outlined in some detail as to method and including evaluation of depth perception, heterophoria and heterotropia, prism adduction and abduction, associated parallel movements, accommodation amplitudes, and angle of convergence, besides the usual quantitative measurements of vision and medical examination of the eyes. As was shown so graphically after the war with reference to the Howard-Dolman test, many of these tests leave much to be desired. Part of our job is to furnish new methods for gaining the needed data.

We now have some type of routine eye testing in the public schools in most areas of the United States. Just recently the State of California passed a compulsory law to this effect. Many ophthalmologists over the country recently received a multiple-choice questionnaire which was designed to gather data for California regarding which examinations should be done and which children should be referred to

an eye specialist. Was referral to be on the basis of visual acuity alone? How much loss? Should fusion be tested? How? Does this constitute reason for referral? And so on.

Before all of you wonder where this discussion is leading us, let's try to find the common meeting ground in all the various things that have been discussed. This is the broad field of measurement of visual acuity. It is this same broad field that, in my opinion, the orthoptist is now being called upon to help cover and, I think, will be increasingly involved in, irrespective of what personal opinions you and I may have regarding it. Granted that most of the time of the orthoptist will probably be spent working with the strabismic child, more and more needs for the accurate, precise, and quantitative determination of binocular visual processes are becoming apparent. I believe that the orthoptist will more and more, in conjunction with the industrial ophthalmologist, get involved with certain specialized industrial screening methods. More and more will the orthoptist come in contact with patients requiring quantitative tests of vision, in some cases involving workman's compensation statutes. More and more will orthoptic tests be made of persons in whom accurate measurement of stereoscopic abilities should affect the desirability to allow them certain privileges, such as piloting an airplane.

The challenge doesn't come all at once; it creeps up on us a little at a time, and too often, I am afraid, we don't recognize it for what it actually is. When one of these slightly unorthodox opportunities comes along, let's all recognize it for the challenge that it is, and realize the genuine fun that can come from many of these situations.

Though I don't believe in advocating courses of action from a purely negative point of view, I believe that we should remember that there are other groups with less background for handling such problems, groups which are figuratively "itching" to get into the show.

In summary, then, may I simply say that I believe there are genuinely broader fields ahead in orthoptics, and that these are primarily concerned with quantitative determination, rather than therapeutics, and that this situation has been brought about by general advances in civilization which require a more definite evaluation of fusional capabilities.

WHAT AN OPHTHALMOLOGIST EXPECTS FROM AN ORTHOPTIST

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I HAVE been in practice for many years and have for some time been interested in the practice of orthoptics as an aid to comfortable vision, apart from its use in connection with the tropias and preoperative and postoperative treatment.

My comments will undoubtedly be critical, and I hope in some ways, helpful. They are the result of association with a number of orthoptists in several towns.

And now, regarding the subject of what an ophthalmologist expects from an orthoptist.

First, may I say that a good orthoptist should be considered as the ophthalmologist's right-hand man, or girl Friday. I believe that the orthoptist should have a free hand in handling the patient and that her advice should be taken by the physician as of considerable importance.

On the other hand, I believe that the orthoptist should recognize the fact that the patient has been referred to her for treatment, and that the patient is essentially, therefore, not her patient but rather that of the doctor referring. I have heard rumors of complaints in the past, to the effect that orthoptists tend to take too much upon themselves when dealing with patients.

It is my feeling that when a physician is doing refractions, he should in all instances include a Maddox rod test for distance and a double-image test for near. Personally, with practically all of my patients, I do a Maddox rod test for distance, a test of amplitude of fusion for far, a test for cyclophoria, a double-image test for near, and a test of amplitude of fusion for near.

In addition to these procedures, I feel that patients in the prepresbyopic stages should have their near point of convergence determined to discover whether or not it has receded beyond the limits of reasonable comfort.

It has been my experience that many patients, probably more of those fitted by optometrists than by physicians, are wearing prisms which act merely as crutches and which tend to increase the deformity rather than reduce it. Many of these patients could be made perfectly comfortable by orthoptic training, and many of them should not have had prisms in the first place. As you all know, practically all of these patients suffer to some extent from suppression. One criticism I would make of orthoptists is that they do not always completely eradicate, or mitigate in so far as possible, the suppression present.

I hate to mention the word *optometrists*, but I have from time to time received patients from optometrists in this city who are doing orthoptics, and I am forced to say that the condition of their patients is sometimes much better than that of some patients who have been treated by orthoptists.

The reason for this, I believe, is that the optometrists continue to treat their patients indefinitely, not for a limited time. I do not support this practice as

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being entirely satisfactory either, because I do believe that in some cases, particularly the tropias, the optometrist tends to overtreat the patient.

Therefore, I now say to you that I feel that an orthoptist should be allowed sufficient time to complete the necessary training of the patient. In a case of tropia the patient's training should continue until his status is optimal for surgical intervention. It has been my observation that when this is done, the surgical results appear to be a great deal better than when the orthoptist merely continues treatment until reasonable fusion has been obtained, then sends the patient in for operation. May I again refer to the optometrist, who usually treats his patient until he has achieved some degree of simultaneous perception, fusion, and, in some instances, even relatively good stereopsis.

Another of the disadvantages of too little orthoptic treatment is that the patient becomes discouraged when his condition returns to that for which he undertook treatment. He concludes that orthoptic treatment has no value whatsoever, whereas in reality he would probably have obtained a great deal of relief had the treatments been continued.

Referring again to the question of the handling of the patient, it is my definite feeling that periodic reports should be given to the doctor. At times these reports are somewhat confusing and should perhaps be more explicit. I know that when I first received reports from Mrs. Wood, I found it a little difficult to understand what an "x" might mean for exophoria. May I say that I personally rely very definitely upon the measurements taken at the Orthoptic Center on surgical patients.

It is true that measurements can be taken in the office, but they are much more accurate when taken with the instruments which the orthoptist uses than with those we use. Therefore, it is, to my mind, extremely important that these measurements be accurate and explicit.

This paper would not be complete if I did not refer to the controversial subject of accommodative spasm. I know that many members of my profession appear to discount this particular entity as being of no importance. Personally, I believe that it is more often present than is generally considered. I have found that it occurs particularly in people doing quantities of close work. I need not stress the point, nor point out to you that our eyes were not intended for close work; therefore, overconvergence, together with overaccommodation, may produce not only a condition of esophoria but also one of accommodative spasm. It is present, in my experience, particularly in teenagers who are emotionally upset and who are, at times, very difficult to treat.

I have had cases of accommodative spasm in which I have had to atropinize the eyes for as long as two to three weeks at a time before I managed to relax them. Often in older people there is some underlying psychological factor which is producing the condition, such as bad home conditions or dissatisfaction with the job that they are doing.

With regard to treatment of accommodative spasm, I believe that divergence exercises are necessary. I have from time to time also used homatropine, in a strength of 0.10 per cent, together with the exercises, in order to allay this spasm.

It is my opinion that the orthoptist should very definitely inform the physician if she finds a condition suggestive of accommodative spasm, and she should ask the physician what treatment he wishes given. I do not propose to go into the details of measure-

ments, etc., which may be found in this entity.

Before summarizing, I should mention a case which I am referring to the Orthoptic Center, and hope will be treated there.

This patient was examined five years ago by a professional colleague of mine, and a very excellent man. She came to me complaining of discomfort, particularly for close work, and difficulty with seeing at a distance. The lenses which she was wearing were −2.00 sph. for the right eye and −1.50 sph. for the left eye. The age of the patient is 31 years and her occupation is close work of some sort, I believe stenography. She complained of frequent headaches with nausea, but with no vomiting. Her symptoms were particularly marked when she was typing. Her eyes burned and she had epiphora but no other eye pain.

Upon refraction with mydriasis she was found to have for the right eye −1.00 sph. −0.25 cyl., axis 180, for the left eye, −0.75 sph. −0.50 cyl., axis 20.

You will notice that the correction for the right eye under mydriasis was just half that of the lenses worn, and that for the left eye was also just half, with an additional correction for astigmatism, which was not in the original prescription.

After the dilatation had subsided, she accepted a correction of −1.50 sph. −0.25 cyl., axis 75 for the right eye and −1.00 sph. −0.50 cyl., axis 20 for the left eye. Muscle-balance for far was orthophoria; for near, 12 degrees of exophoria. Amplitude of fusion for near was 16 degrees base out, with diplopia, right eye deviating. The near point of convergence was a 200 mm., the right eye deviating with diplopia at this point.

This, I believe, is probably a case of accommodative spasm. There is no other reason that I can suggest why the overcorrection was made. It must be noted that the patient at the time of the original examination was 26 years old. She seemed to be an emotional person.

The reduction in error at this time, although still not complete, is the result, no doubt, of the relaxation of the ciliary muscles. Orthoptic training has been ordered, the diagnosis being divergence excess. Confirmation of this diagnosis is anticipated.

Some time ago a student came to me with a visual acuity of 20/80 in both eyes. With mydriasis his vision was normal, and he had no visual error whatsoever. His complaints were headache and difficulty with distance vision. He accepted a minus correction without mydriasis. This case is another example of accommodative spasm.

Now to summarize this paper: May I say again, that I believe that the orthoptist should be the girl Friday of the ophthalmologist. Orthoptists should not, however, assume that because a patient is referred to them, the patient is theirs. Rather, the patient has been loaned to them until such time as treatment has been completed. They should have full control of the patient while he is under their care.

The ophthalmologist should be prepared to accept the recommendations of the orthoptist, but the patient should be returned to the ophthalmologist in due course, when treatment has been completed. Training should not be discontinued until the time has come when you are positive that nothing further can be done for the patient, whether he is to be operated on or not.

It is my feeling that it should be the forte of the orthoptist to sell orthoptics, both to the physician and to the patient.

I hate to refer to optometry again, but I must point out to you that I know of three men in optometry who are doing very well with orthoptic training, and who are charging exorbitant prices for their work. All these men are doing a very large volume of work, and they are definite danger to the orthoptist. For this reason I say again that it should be very definitely the forte of the orthoptist to sell orthoptics whenever possible.

TECHNICIAN-SPECIAL EDUCATION OR GENERAL EDUCATION

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How many lost children are there in our schools? How many are actually learning and able to take full advantage of the program offered? Every teacher is concerned about the child who is not making progress, but how often does she reach the root of the trouble and discover why he is not learning?

Too often after a child has experienced failure time and again, we find that he has never seen well enough to carry on work designed for the so-called normal child. By the time this is discovered, he already has acquired the stigma of a slow learner. He has built up a defense against this and has developed a pattern of thought processes that may never be broken or will take years of skillful handling and guidance to break. So, I ask you to consider carefully your decisions when you say, "Let him try the regular class as long as he can," or "Allow him to stay in the regular class until he shows he can't carry the work." Are you saying, "Let's wait until he becomes a failure—then we will place him in a special class"? Perhaps you mean to catch him at a time when his work is just beginning to slip, but the parents, not welcoming the prospects of a special class, will often interpret your remark as, "He may stay in regular class as long as the school will permit." The classroom teacher may not sense the effects of failure until the child shows signs of extreme behavior and becomes an attendance problem.

This situation demonstrates a need which both teachers and orthoptists must be aware of; that is to improve our communication. I cannot propose a solution, but will leave the thought with you. You give your diagnosis and directions for treatment to the parents at the time you see the child. You are not anxious to make the parent overconcerned, and rightly so. Therefore, you say to the parent that everything is going to be all right, or that the child shouldn't be under too much pressure, or some similar words of advice. There are three groups of extremes among parents: those who feel that the youngster may go blind any day, those who feel that there is absolutely nothing wrong and the child should see, and those who get so upset that they know nothing of what you say.

Then we have three groups of extremes among the teachers: those who are very much concerned and feel that the child should not be expected to do any work, those who feel that every child in the class should be treated alike and a child who has anything wrong with his vision belongs in a special class, and those who are unaware of anything that happens outside the classroom—so what!

You and I come between these groups of people if we are truly interested in what happens to these children. Of course you can take the attitude that the children came to you for a visual examination and you gave it—so that's it. I can say that my obliga-

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tion is to help parents and children make adjustments and secure registrations in the special class—so if a child is not eligible for a special class he is not my problem. But, I don't think any of us feel that way; we want to help all children. I want parents and children to have a positive feeling about their eye examinations because I hope they will return for another one. If I can do anything to protect the vision the child has, I want to do that, and I am sure you do too.

I would suggest that you try to evaluate the parent while you are seeing the child. Also, if the anticipated treatment might attract attention to the child, some one in the school should be contacted in order that interpretation be made. For example, let's consider a simple thing like an eye patch. In one of our schools we had a child who required an eye patch. The child came to school with no explanation as to why or how long the patch was to be worn. The teacher did not know either, so no explanation was made to the class. I happened to stop in the building, and everyone, from the custodian to the children, was upset because this child "had her eye bandaged." "No doctor would ever do that to a child of mine" – and so on and on. In another case, the teacher was contacted, the process was explained, and the teacher, in turn, told the class about eye muscles and how they can be strengthened. A good class discussion led to intelligent acceptance of the patch, and the child involved felt no pressure. In the first case the patch caused adverse comment; in the other case it offered a basis for a good lesson and acceptance.

Visual handicaps of any kind jeopardize the individual's chances of making good social adjustment. Too often our full efforts are centered around the individual involved or in helping the parents to manage their feelings about the child and his handicap. The social adjustment of the child is an important phase of any approach to prevention or correction, whether it involves the placing of glasses on a child or surgical correction of his defect. We are always dealing with two handicaps—one physical and the other emotional.

Personality arises as a result of the interaction of an individual and his environment. The major requisites for the development of a child's personality are the calm, accepting love of his parents and full social contacts with his contemporaries within the limitations of his basic abilities. We must always consider the parent's reaction to a child with a congenital visual defect. How will a parent react when he finds himself responsible for the care of a child who has developed a visual handicap through accident or illness? No ready-made stock reply will answer these questions.

Naturally, all parents think of their children as being superior to others. A handicapped child is a threat to any parent who is not emotionally mature. Usually the parents react by being overprotective. Some reject the child. Some develop feelings of guilt and punishment, and try to make it up to the child by overindulging him. Under these conditions a child finds it impossible to develop self-reliance, confidence and security.

Rejection may be overt, but most often it is displayed as inconsistent management and expectations of behavior far exceeding the child's ability. The child then manifests tensions, bitterness, frustration, and anxieties. As a result, he has two handicaps rather than one. That is why we teachers may ask the orthoptist what reaction the child and the parent have toward the handicap. None of those adverse reactions is conducive to learning. We

are not prying for information which you might feel you cannot ethically give.

Being able to read is most important to the child if he is to be successful in school. The first-grade child who doesn't learn to read is looked upon as being different by the other children in his class. His parents become overly concerned, and the school is supposed to give the parents an answer to "Why is my child not learning to read?" Our first premise is to rule out all physical causes. Then, a psychological examination may be recommended. Many parents would far rather find a physical cause for the lack of academic success than accept the diagnosis of low intelligence.

Many factors of an intelligence test depend on visual acuity. When we test a child who shows symptoms of visual difficulty, we request further visual tests; the school nurse may do further screening, or if the possibility of visual problems is brought out to the parents at the time the test is interpreted to them, they may take the child to the doctor. In any event, it is possible that parents will bring their child to the ophthalmologist for examination during this period during which the child's lack of academic success is being investigated. All we ask is that you study the parent's reactions, and contact the principal, the teacher, the school nurse, or the counselor if you are interested in the position the school has taken in the case. Remember, if there is need, the school will contact you either through a worker or by letter, so do not interpret the position of the school through the parent alone. Remember too, the child, who is most important to his parents, is a failure in the school. I think we teachers and orthoptists can maintain better relations if we do not judge each other through dissatisfied parents.

Out of the child's inability to read grows another situation. Because of his poor grades, he begins to rub his eyes and complain of headaches; he may even say that he has spots before his eyes or that he has periods of blackouts. We can say to each other that we think these are psychosomatic manifestations, but we are not willing to let it go at that. We recommend a visual examination. If the child's vision is found not to be the source of his trouble, the parents are often disappointed, and sometimes the ophthalmologist is critical when a child is referred to him when it isn't necessary. But, who is the judge? In such a case I prefer to explain the symptoms to the parents, ask them to observe the child at home while he is under no reading pressure and note whether the symptoms persist. However, if they become overly concerned and have the child's eyes examined, it is their decision.

I feel that public concern for visual care and prevention of blindness has been on too casual a basis. To most people, *failing sight* suggests a matter of changing glasses or the thought that we must do all we can to help the blind. We need more education on how to recognize early symptoms of eye trouble. How often do we read an article for the promotion of better vision? Once in a great while we see an article about a corneal transplant or the eye bank, but we do not have the public really conscious of the care of the eyes. Public education along this line could become as effective as is that regarding prevention of tuberculosis, heart care, and recognition of cancer. Moreover, since much work dealing with the care of the eyes is done in an eye clinic, people need to know something of how a clinic operates (the waiting periods, the change of doctors) and how it can best serve their need.

There is a need for a general understanding of vision: the effect of disease and eye strain, the decrease of night vision, resistance to glare, and the normal changes of vision with age. With this information, people would better understand what to expect of their eyes.

I think you will agree with me that we need better visual screening devices for use in our schools. In 1948-49, the public schools of St. Louis tested the vision of all their first and sixth graders. Their screening devices included touch judgment, the Snellen test, the Massachusetts vision test, the Keystone View Telebinocular, the Bausch and Lomb Ortho-Rater test, the American Optical Company Sight-Screener test and the near vision test. A complete eye examination was given each child by an ophthalmologist. One ophthalmologist evaluated all the test findings and recorded a clinical judgment of "refer" or "nonrefer." No one test or any combination of tests offered a reliable means of determining those children who should be referred for further examination. The doctors who took part in the study agreed that further research in screening was needed.

According to the 1949-50 statistics, we had twenty-eight million children in our schools. Five thousand, five hundred and sixty-five of those children were blind; that is about one in five thousand. Seventy-six per cent of the blind children were between the ages of 3 and 25. Ninety-three per cent were between 5 and 19. In 82 per cent of these cases the onset of blindness took place under 5 years of age. Sixty per cent were blind at birth. Hence, a problem is raised regarding the prenatal factors that contribute to blindness. What factors have a harmful effect on the development of the eyes? These are things which we need to teach to our high school students in our health classes.

What can be done in order to get better vision tests as a part of the examination for drivers' licenses? An article in Esquire written in 1952 estimated that twenty million motorists at that time had below-average eyesight, and three million of them didn't know of their defect. Visual competence for driving the fast, powerful cars of today involves more than just 20/20 vision.

What can be done for the children in the rural areas where there are no special services in the schools? It has been estimated that only one out of seven children in the state who need special class placement is living where such service is available.

You are going to have many more referrals from parents since all the interest has been aroused over children not learning to read. Newspapers and magazines are playing up the problem more and more. You and I know that reading involves far more than good vision and a good teacher. Most parents do not understand that fact; therefore, we each have a big problem of interpretation if we are going to work together.

In summary, I hope that you see the close relationship between the work of the school representatives and your own. What we teach depends much on what you find out in your research work. We each need to do more to keep the public informed. If you could share some of your fine articles, written for your periodicals, with teachers' magazines and we in turn could send some of ours to your publications, we would probably have a better understanding of each other. You read about your profession and we read about ours; and neither reads too much about the other. The more I understand about you and your work, the more service I can be to the boys and girls in the Bloomington Public Schools.

STRABISMUS UNDER FOUR YEARS

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The importance of the early diagnosis and treatment of strabismus in the infant has been stressed by leading ophthalmologists for many years. Fortunately these teachings are bearing fruit as squinting infants are being brought to the ophthalmologist ever earlier in their disability.

It is interesting to note that the onset of squint in 75 to 90 per cent of cases, according to various estimates, [1, 6, 7, 9] occurs before the patient is four years old. The realization by ophthalmologists and other physicians that the majority of squints do develop very early in life is reflected by the early age of the patients seen in the muscle clinic and in the operating room.

The earlier diagnosis of strabismus shifts the emphasis of management and treatment from the 4-to-8-year-old group of patients, with all of their sensory and motor complications of misalignment, to the more pliable 6-month-to-3-year-old group.

To cope with this younger age group there is a definite need for emphasis on the development of special orthoptic techniques suited to the group. In the past the ophthalmologist has tended to be the sole therapist for these young children. However, the study and understanding of the variables of each infant with strabismus, as well as the development of the cooperative efforts of both the mother and infant, require time and skill that can best be supplied by the orthoptist.

To better understand the management of early strabismus the ophthalmologist and the orthoptist must become well acquainted with the physiologic components required for the development of binocular single vision.

The normal development of binocular single vision depends upon the perfection of the interrelationship of fixation, visual acuity and fusion. Fixation is fleetingly evident when the child is only 2 weeks old, and by the time he is 6 weeks old massive objects may be fixed for many seconds. By the age of 5 months an exact simultaneous binocular fixation ability has developed sufficiently for the child to follow a moving object and reach for it. As reflex fixation movements develop and become more firmly established, the eyes wander less and remain parallel most of the time.[3]

The improvement of the fixation reflex during the early months of an infant's life is closely related to an improving visual acuity. Visual acuity, which at 5 months is near 20/2000, approaches 20/1000 at 6 months, and 20/200 at 9 months. This gradual development continues so that by the time he is 2 years old visual acuity approaches 20/40.[4] While fixation improves visual acuity, visual acuity also improves the quality of fixation. This development of better acuity and better fixation takes place at the same time and in each eye.[3]

The fixation reflex is monocular, and an image need fall on the retina of only one eye for the adjustment of fixation to take place. The innervation required for moving the fixing eye to a fixation point is transmitted to both eyes so that they then act as a single

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unit. However, there is always some deviation of the nonfixing eye; so whenever the images seen by the two eyes are not on corresponding areas of the retinas, a reflex adjustment is made allowing the image to fall on the proper retinal area and making binocular fixation possible. The purpose of this fusion reflex is to correct any imperfections in the coordination of the two eyes.[3] The anatomical basis of binocular single vision is complete by the time the child is 6 months old; the reflex is refined before his second birthday and fixed when he is 5.

The fusion reflex by which we maintain binocular vision has a sensory component and a motor component. The sensory component requires a nearly equal or symmetrical image to the two eyes. The motor component is dependent upon a satisfactory sensory stimulus, plus the transmission of the proper motor response to intact extraocular muscles.[2]

If from early infancy some obstacle prevents the use of an eye, a good fixation reflex will not develop; visual acuity will be arrested, and the fusion reflex will not appear. The result of impaired vision of one eye is often strabismus, and when both eyes are affected, the result is nystagmus. The obstacles to the development of a normal fusion reflex may be optical, sensory, motor or central. Some of the more common optical obstacles are refractive errors, anisometropia, opacities of the cornea, congenital cataract, and vitreous opacities. The sensory obstacles that may be encountered are congenital demyelination of the optic pathways and anomalies and inflammatory processes of the retina. Some of the motor obstacles are trauma affecting the globe, orbit, and adnexa; muscle anomalies, such as Duanes' syndrome and strabismus fixus; and injury to the motor nerves or to their nuclei. Central obstacles affecting the cortical areas of the occipital and frontal lobes may be the result of physical or mental trauma or generalized infections, such as the acute contagious disease of childhood.

The management of strabismus resulting from anatomical obstacles to the development of binocular vision usually consists of cosmetic surgery in a few selected cases. There is very little opportunity for a functional improvement or for the development of binocular vision in these children.

A discussion of the management of any condition presupposes a knowledge of physiology and underlying factors in the condition. Since strabismus in children is largely innervational, a discussion of this subject must also include some elucidation of the vergences.

It seems to be generally accepted that there is an active convergence mechanism, but there are widely different opinions as to the presence of an active divergent mechanism. It is thought that convergence is controlled by a cortical and a subcortical mechanism. The subcortical convergence mechanism is believed to be constantly bombarded by stimuli from many areas which, unless suppressed, would result in a constant and exaggerated convergent position.[2] To control the tendency for excessive convergence, a hypothetical cortical mechanism is assumed to inhibit the constant overactivity of the convergence mechanism and thus to maintain binocular vision. Release from the inhibiting power of the cortical center by anoxia, alcohol or emotional instability causes an exaggerated convergence and a resultant esotropia.[2]

If the eyes do assume a heterotropic position, there is resultant visual confusion and diplopia. To overcome this confusion and diplopia, suppression in either one or both eyes occurs. If the heterotropia persists, the suppression is followed by amblyopia; the amblyo-

pia, by the development of abnormal retinal correspondence; and finally, the abnormal retinal correspondence, by an eccentric fixation. The whole chain of sensory complications may not be evident in any one case, but each successive complication is an adjustment to the preceding one.[3] The motor component of the visiomotor apparatus develops complications to the tropia such as a pseudoparesis followed by hyperactivity and, finally, fibrosis of the antagonistic muscle.

A great deal of emphasis has been placed upon the treatment required to correct the sensory and motor complications in a child 3 years of age or older so that binocular single vision can be re-established. In considering the treatment of children from infancy to the age of 3 years who are just beginning to manifest a squint, the emphasis needs to be shifted from the treatment of established complications of heterotropia to the prevention of the development of these complications. The pliable infant responds rapidly to thoughtful, well-executed management. Because there are no deeply ingrained complications, the opportunities for the development of binocular single vision are greatly enhanced.

In this discussion of the management of innervational strabismus, Costenbader's classification[2] will be used because of its sound basis in correct physiologic principles.

Innervational strabismus may be either accommodative or nonaccommodative. If the esotropia is accommodative, it may be either refractive, hypoaccommodative or hyperkinetic. The refractive type is characterized by a hyperopia of three to four diopters in infants aged 12 months to 4 years. The excessive innervation required for accommodation exaggerates convergence so that an esotropia results. The wearing of lenses with full refractive correction usually results in esophoria or orthophoria. When suppression or amblyopia has developed before the child is first examined by the ophthalmologist, treatment by patching and simple light stimulation results in a rapid recovery of vision and fixation.

The hypoaccommodative type of esotropia is the result of excessive stimulation of a weakened accommodation mechanism with a resultant overconvergence. The differential point in separating this type from the other types of accommodative esotropia is the remote punctum proximum. A full refractive correction utilizing bifocals lessens the accommodative effort, but there is frequently a residual esotropia for which an operation is contraindicated. As esotropia of this type gradually decreases with time, it becomes the responsibility of the orthoptist to prevent the development of sensory complications that would preclude binocular single vision.

The hyperkinetic type of acommodative estropia may appear between birth and 4 years of age in the bright, nervous, unstable child. Characteristically, the esotropia varies from orthophoria to 30 or 40 prism diopters at various examinations. The cause seems to be a weak cortical inhibition of the convergence center so that even a minimal accommodative effort excites an exaggerated convergence reflex. The treatment consists of full refractive correction with bifocals and the prevention of sensory complications. As emotional stability in these children develops, the esotropia improves remarkably.

The nonaccommodative esotropias may be of either the tonic or the mechanical type. The congenital tonic type of esotropia develops within the first six months of the child's life. The large esotropia is usually 60 to 80 prism diopters and is little influenced by correction of the hyperopia. Significantly, the eyes straighten under gen-

eral anesthesia. This type of esotropia is probably due to a defect of the cortical inhibitory mechanism. The cortical area may not have developed normally, or there may have been damage to the area during fetal development or at birth. The causative factor is probably not congenital paresis of the lateral rectus muscles, as is often suggested, although there may be limitation of abduction of the eyes. The primary treatment in these cases is surgical recession of the medial rectus muscles as soon as there is evidence that the esotropia is no longer increasing. This is usually when the child is between 12 and 18 months of age. During the waiting period the prevention of sensory and motor complications is of extreme importance.

There is also a tonic nonaccommodative esotropia of later onset. It may appear suddenly between the ages of 2 and 6 in hyperactive, bright children. This type is also apparently due to an inadequate cortical inhibition of the convergence mechanism, with the resultant overaction of the medial rectus muscles.

The mechanical type of nonaccommodative esotropia usually develops in infancy. The esotropia is large and constant, and it is associated with only a small amount of hyperopia. It is differentiated from the tonic type in that the eyes remain convergent under general anesthesia. After repeated examinations a congenital paresis of the lateral rectus muscles, an anomalous muscle insertion, or a congenital malformation of muscle structure may be found. Infants with this defect develop suppression, amblyopia, and abnormal retinal correspondence early and deeply. Management consists of combating the development of sensory complications by orthoptics and performing the surgical operation indicated in each case as soon as is reasonable.

Although Costenbader's classification[2] of innervational strabismus is of inestimable value in the understanding and the management of these cases, most of the children who come to the clinic have components of both accommodative and nonaccommodative esotropia. Frequently their condition is further complicated by the development of secondary sensory and motor complications. For example, a child with an accommodative refractive esotropia will have superimposed nonaccommodative esotropia of the congenital tonic type. With careful analysis and study, each of the components can be separated and their relationship to each other determined so that the proper surgical, refractive and orthoptic procedures can be carried out.

Innervational exotropia has an onset as early in life as that of esotropia, but in the majority of these cases the deviation is intermittent until the child is 3 years of age or older. One important exception is the divergence excess of early infancy. The exotropia is manifest at birth or shortly thereafter and may have a large degree of deviation. The initial management of these cases is orthoptic, although in exotropia the severe sensory complications of esotropia do not usually develop. Surgical intervention is indicated when secondary convergence insufficiency appears and progresses or when there is an exotropia of 20 prism diopters or more at distance.

This brief sketch of some of the causes and some of the indications for the management of strabismus in infants is presented in the hope of stimulating interest in the development of binocular single vision. The early recognition of strabismus means that an increasingly large number of children with this disorder will be brought to the ophthalmologist at an age in which binocular reflexes are developing. The development of special orthoptic techniques is needed for the treatment of

these patients. However, the lability of the ocular reflexes and the pliability of the infant make the results most gratifying.

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DIPLOPIA

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WHY should I choose to discuss diplopia? Because I think training the child to use both eyes is one of the most important techniques in the treatment of squint – and one of the least stressed. More and more I see patients who have been treated by other reputable technicians – patients who have been discharged as cured – who still squint intermittently. Maybe you've seen them too. They are usually patients with intermittent exotropia who have excellent convergence, fair fusion, and no diplopia. Are they aware of their squint? No. Does it bother them to squint? No. These, I think, are the main purposes of teaching diplopia: (1) to make the patient aware of having two eyes and of their not working together; and (2) to make the patient uncomfortable when he does not fuse.

It may sound, as I am talking, as though I were giving a class on suppression rather than on diplopia. But what is diplopia training if not the breaking up of peripheral suppression? And in order to prescribe the correct diplopia exercise, the type of suppression must be diagnosed. My main emphasis during this discussion will be on regular diplopia, but I will also include both physiological diplopia and monocular diplopia (or, more correctly, binocular triplopia). We shall consider all three types from the standpoints of both diagnostic examination and treatment.

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DIAGNOSTIC EXAMINATION

For whom: All patients with strabismus
Question: What information is provided by tests for diplopia?

We do an examination to answer questions about a patient. What is wrong with this patient? What is his binocular status? Can orthoptics help him? Let us take some specific questions which can be answered by testing for diplopia: (1) How deep is this patient's suppression? (2) If it is possible to elicit diplopia, how far apart are the two images, i. e., does the patient have normal or anomalous retinal correspondence? (3) How large is the area of suppression? (4) Does the patient have a blindspot mechanism? These questions cannot necessarily be answered by consideration of the length of time the squint has been present nor by determination of the visual acuity. It is not unusual to find a patient with long-established squint who has learned that a certain amount of suppression is enough for him to escape diplopia. Suppression is an active process, and therefore requires some effort. We often find patients who, after a short period of occlusion with little improvement in visual acuity (macula), have spontaneous diplopia (periphery). So we must answer these questions by specific tests.

For whom: All patients with strabismus
Question: How deep is the suppression?

How can we measure the depth of the existing suppression? By discovering what is required to overcome the suppression. I do this by means of layers of red kodaloid over the *fixing* eye. I realize that this is contrary to the

practice of the authorities on this subject. However, I believe that my reasons are valid, and I seem to get better results by covering the fixing eye with red than by covering the nonfixing eye. In my diagnostic work, as well as during treatment, I want diplopia obtained with the usually fixing eye fixing, because that is the patient's normal way of looking. Moreover, children, particularly small ones, would rather look at a "pretty" light than a plain white one. Also, by darkening the image in the usually fixing eye, the peripheral image in the nonfixing eye becomes more easily distinguishable. Consequently, I use as many pieces of red kodaloid ("reds") over the fixing eye as are necessary to obtain diplopia. (Wasn't it Alice McPhail who hit upon the idea of a red-glass bar? It is perfect for this sort of thing. It is built like a prism bar, only instead of prisms it has an increasing number of layers of red kodaloid, so that at the bottom of the bar the red is very dark, while at the top it is very light.) I note on the patient's record the number of reds needed, because that tells me how deep is the suppression: if it takes five reds, the suppression is deep; if only two are necessary, the suppression is not deep. If, even with five or six reds over the fixing eye, no diplopia is obtained, the prognosis for rapid breaking up of suppression is not good.

For whom: All patients with strabismus
Question: What is the retinal correspondence?

How far apart are the two images which the patient sees? If the patient has not achieved diplopia, can he do so by use of prisms (base-out prisms in esotropia or base-in prisms in exotropia)? If so, does the separation coincide with the patient's angle of squint? In making diagnostic tests I usually measure this separation with prisms. It can also be measured by distance.

It was Dr. Burian who first made me realize why we had so many discrepancies in our tests for anomalous retinal correspondence (ARC). He pointed out that tests most resembling casual seeing are the first to demonstrate ARC and are also the last to indicate achievement of normal function during treatment. Thus, on the troposcope during treatment, the angles are more likely to correspond with first-grade targets (we are not presented with dissimilar targets for the two eyes in casual seeing) than with second-grade targets. Likewise, the result of an afterimage test (unlike anything we are called upon to use in everyday life) is often normal when results of other tests are anomalous. The result of the afterimage test is more easily made normal with thought than are the angles on the troposcope.

What does this have to do with diplopia? It is immensely important in a red-glass test. The red-glass diplopia test, when prisms are used ("Tell me when the red jumps to the other side") may demonstrate normal retinal correspondence (NRC). At the same time, when done without prisms ("Show me how far apart those two lights are") ARC may be demonstrated, since prisms create one more obstacle not encountered in everyday seeing. So there are two ways to measure the separation of the images, and by using both measurements we get an indication of the depth of the ARC.

1. During the initial visit I usually measure the separation of images with prisms. That is, if a convergent squint is present, I use base-out prisms in increasing strength until the images are seen to cross over to the other side (crossed diplopia). This crossing place is noted on the record. If this prism measurement does not come within a few prism diopters of the prism and cover findings, I write "ARC" promi-

nently on the records, so it is easily seen during subsequent visits.

2. If ARC has been detected by means of other tests and yet the red-glass test done with prisms shows NRC (which often happens after a few orthoptic treatments), I then do the test without prisms. A prism diopter, by definition, displaces a ray of light one centimeter at a distance of one meter. Therefore, at 20 feet (6 meters) a ray of light will be displaced 6 cm. by 1△, 12 cm. by 2△, etc. I use as many reds as are necessary to obtain diplopia; then I ask the patient to walk up to the tangent screen and show me where the white light was and where the red light was. If the patient has an esotropia of 30△, the separation should be about 180 cm. (nearly 2 yards). If the test is done at 13 in. (⅓ meter), great care should be taken that the fixation distance is measured accurately, since a few inches backward or forward makes a big difference in the amount of separation of the images.

For whom: Patients with suppression and normal retinal correspondence
Question: How long is the area of suppression?

When diplopia was obtained on the previous tests, was it at the patient's usual angle of squint or, as I watched ever so carefully, did the patient straighten his eyes slightly or squint just a bit more to avoid his usual area of suppression? There are three good ways of measuring the size of the area of suppression: with prisms, with the major amblyoscope, and with a device such as the Travers mirror.

1. Let us take the first method, with prisms. Suppose we have a patient with an exotropia of 20△ at distance and needing three layers of red kodaloid over the fixing eye in order to obtain diplopia. I place *one* red over the fixing eye. The image of the light is now falling on a peripheral area in the *nasal* portion of the retina of the nonfixing eye. For the sake of convenience, let's call this portion of the retina "Area X." The patient is suppressing the image of the light from Area X, since we are using only red over the fixing eye. Now I use my prism bar base in on the nonfixing eye, which throws the image of the light on an area of the nasal portion of the retina farther from the macula than Area X. The prism bar (or rotary prisms) cannot be changed rapidly, as this would give the impression of a moving object, which is not easily suppressed. So with the prism bar held base in, I stop at each change of prism and ask the patient if he sees two lights. I make sure that he does not alternate but fixes continually with the eye beneath the red. A note is then made on the record of what, if any, prism base in gave him diplopia. The same thing is then repeated with the prisms base out. The entire test is then done at near.

2. To measure the area of supression on the major amblyoscope, I use small, undetailed first-grade or second-grade targets. The lack of detail is important with small children, because to them "to see something" is synonymous with "to see clearly," or at least clearly enough to identify the image. For a deeper suppression I use a first-grade target; for a less deep suppression, a second-grade target, since a second-grade target more nearly resembles objects in everyday seeing and consequently is more easily suppressed. Beginning at the angle of squint, I slowly move the target before the suppressing eye more base in, noting when the target disappears and when, if at all, it reappears and again disappears. Then I repeat the process, moving the target more base out from the angle of squint. As with prisms, this test is done at distance and at near, using -3.00 spheres for near. Often in a patient

with accommodative or partially accommodative squint, the area or areas of suppression change or increase with an increased accommodation.

3. The Travers mirror is a small mirror mounted on a calibrated scale in such a way that it rests at an angle against the patient's nose while he sits with his head in a head rest facing a tangent screen one meter distant. On the wall in back of and to each side of the patient I have placed stickers. I happen to have a booth in my office which is slightly over one meter wide. The stickers are used as fixation objects. If the mirror is angled before the right eye, the sticker on the right side of the patient can be seen in the mirror; if the mirror is angled before the left eye, the sticker on the left side can be seen in the mirror. The mirror, for this test, is adjusted before the fixing eye at zero on the scale. As the patient fixes on the sticker, a regular central field examination of the other eye is made; that is, a white-headed pin, 1 or 2 mm. in diameter, is moved about and the areas where it is not visible are marked out on the screen. The patient must be watched closely, as in any central field examination, so that he does not change his fixation but maintains it constantly on the sticker on the wall. As stated before, the pin must be moved slowly in order to delineate the patient's usual areas of suppression.

Results of these three tests, done intermittently during the course of treatment, will give the orthoptist an idea of how the diplopia training and the alleviation of suppression are progressing.

For whom: Suspected cases of blindspot mechanism

Question: How can we detect cases of blindspot mechanism?

With patients in whom I suspect blindspot mechanism, I use a Travers mirror. What makes me suspect that the patient is using his blindspot instead of suppression? (1) The esotropia stays nearly constant between 25△ and 35△. (2) The patient has normal retinal correspondence. (3) Diplopia cannot be attained at the patient's usual angle of squint, but it is easily elicited at angles slightly greater or slightly less than his usual angle of squint. Why do I bring this up in a discussion of diplopia? I am not going to talk about treatment for patients with blindspot mechanism; but we must be able to recognize such patients, since we cannot teach them diplopia. The test is done in one of two ways; I sometimes use one method, sometimes the other.

1. The Travers mirror is adjusted at zero on the calibrated scale, so that the fixation object (that seen in the mirror) would, by a pair of normal eyes, be superimposed upon the center of the tangent screen. If the patient has a blindspot mechanism, his squint will be of such a degree that the fixation sticker seen in the mirror will be superimposed upon the circle on the tangent screen which represents the position of the normal blindspot in monocular fields. At the same time, the center of the tangent screen will fall on the patient's blindspot and will not be seen.

2. The second method of detecting a blindspot mechanism with the Travers mirror is very much like getting the subjective angle on the troposcope. This is a very unscientific method, but it works. The patient is allowed to adjust the mirror so that he sees the sticker (the image in the mirror) superimposed upon the center of the tangent screen. Then I put my head on the head rest, and if, at the position the patient has set the mirror, I see the sticker superimposed upon one of the blindspot circles, I suspect a blindspot mechanism, at least at the one meter distance.

For whom: Patients with anomalous retinal correspondence
Question: Can monocular diplopia be elicited?

Monocular diplopia, as used in orthoptics, has created much controversy. I have always maintained that when monocular diplopia is present, both images are formed on the same set of retinal elements, but are projected anomalously and normally at the same time. I could get very little support from others for my belief, so I began to ask some specific questions. (1) What size must an object be to create a macular image? (2) If that image is giving off light rays in all directions, why does it fall on one part of the retina only, in a normal eye? (3) When monocular diplopia is present, is the anomalous image blurry? The following answers will clarify some of these problems:

(1) Persons more learned in mathematics than I have said that on the troposcope the image of a 5 mm. test object is macular. If, then, a 5 mm. clown is seen and easily recognized in both images of monocular diplopia, are not both images macular? (2) The optical apparatus of the eye (with a correcting lens if required) brings all rays of light from one image to a single focus on one part of the retina only. How then could it suddenly, in ARC, bring the image of a single 5 mm. clown to a focus on two separate portions of the retina? (We are assuming, of course, that our orthoptic patients have no ocular disease.) (3) By carefully questioning some of my older patients, I find that the two images present in the same eye are of equal clarity, though of different intensity. They report that as soon as the correct image appears, the incorrect image becomes dimmer. This monocular diplopia, then, at the objective angle of squint, consists of two macular images—one being projected normally and one being projected abnormally.

What does the presence of monocular diplopia mean to us diagnostically? Well, obviously it means that ARC and NRC are present coincidentally. That, of course, means that the prognosis for breaking up the ARC is good.

For whom: Patients with phoria and suspected esotropia
Question: Is a squint present intermittently?

Consider now a patient who is sent to us because his mother has noted (or thought she noted) an intermittent esotropia, which the doctor has been unable to elicit. There are two good ways to know whether such a squint is present. Since suppression is an active process to escape diplopia in strabismus, there should be no area of suppression if there is no squint.

1. One way of discovering whether a squint is present intermittently is by using base-in or base-out prisms in an effort to demonstrate a suppression area. Suppose we have a patient who may have an intermittent esotropia. If there is no area of suppression in the nasal portion of either retina, an intermittent esotropia is extremely unlikely. I use a prism bar, beginning with the very first prism, and measure the patient's divergence amplitude, watching his eyes very carefully for the limit of that amplitude. If, at the limit, diplopia is spontaneous, I continue with the prism bar (throwing light rays farther and farther from the macula), stopping at each increase in diopters to ask about the presence or absence of diplopia. There may be a place on the bar, for example between 10△ and 15△ base in, at which no diplopia is elicited, but below and above which diplopia occurs. In that case I would assume that the patient occasionally had an esotropia of 10△ to 15△. With a patient who may

have an exotropia I use the same procedure, but with base-out prisms. The test is, of course, made both at distance and at near.

2. A second way of discovering whether squint is present intermittently is by utilizing physiological diplopia. This should be done at near especially in cases of esotropia, because an intermittent esotropia is most likely to occur at near, and as I said before, suppression may be present when the eyes are accommodating and absent when they are not. Furthermore, diplopia is such a peculiar phenomenon that it also may be present with accommodation and absent without it. I stick a blue star in the middle of a pocket mirror and a red star on the patient's nose. When the patient looks at the blue star on the mirror (the closer image) with the mirror at arm's length, he should be able to see two red stars on his nose (actually two noses). He looks at the star on the mirror, since then the image of the red "nose star" falls on nasal portions of the two retinas, the nasal areas being our main point of interest in suspected esotropia. When he can see one "mirror star" and two "nose stars," the mirror is brought from arm's length to the near point of convergence. If one of the stars continually disappears in about the same place each time, or if physiological diplopia was not elicited, I suspect that the patient may really have an intermittent esotropia, else why would he have built up an area of suppression?

For whom: All patients
Question: Which tests should be done on the first visit?

Must all of these tests be done during the first visit? Obviously not, unless you are going to keep a patient working two hours. But these tests must be done before an estimation of duration of treatment can be given either to the doctor or to the parents. That is why, when I was working in Chicago, we insisted on a series of diagnostic visits, rather than a single diagnostic visit, for all patients, except in rare cases. Actually, during the first visit I always check the separation of the images (ARC and NRC) by means of prisms. If the child is very cooperative, the depth of the suppression is tested with as many layers of red kodaloid as are necessary. The measurement of the area of suppression is usually left until the first of a series of treatments. If squint or a large phoria is suspected, a test for physiological diplopia is made during the first visit.

For whom: All patients
Question: What about pseudodiplopia?

While we are considering diagnostic visits, I should like to mention a response to testing which I have noted occasionally but which I have usually blamed on children's answers. It occurred not long ago with an adult patient of mine who had an amblyopia. I have had few amblyopic adults and so have not had the sensation explained to me before. This man had not complained of diplopia until we were going through the various tests; then he said he saw two lights with his amblyopic eye. It took a good deal of questioning and groping around on my part before we realized that because of his very small central scotoma, he did not see the very center of the light but did see the portion of the light above and below the center. This gave him the impression of vertical monocular diplopia. As his vision improved, the sensation disappeared. I suppose this might be called a "pseudodiplopia." At any rate, I am more careful now about doubting children who claim diplopia when the good eye is covered.

TREATMENT

To whom shall we teach diplopia and why? I teach it to all children and to a few adults who have NRC and either intermittent or constant squint. Why? First, because these patients are coming to me to learn to use two eyes together; therefore they must be made aware of having two eyes. Secondly, they must be made aware of their squint and must be made uncomfortable when squinting. All the fusion training possible will not entirely dispel a squint if the patient is still comfortable while squinting. If he does not have a good sound recognition of diplopia, he cannot always tell when his eyes are straight and when crossed, and the technician cannot be sure whether his eyes can or cannot be straightened without a surgical operation. If he is unable spontaneously to straighten his eyes, even after learning a troublesome diplopia, occlusion is instituted until surgery can help him out. Then, if the operation does not exactly straighten his eyes (and how often it doesn't!), with a good foundation of diplopia that patient can often fuse the two images postoperatively although he couldn't preoperatively. He can realize his own progress.

I teach diplopia also to patients with ARC who can, with concentration, appreciate a normal separation of images, whose answers are reliable, and who are mature enough to understand what they are doing and why.

In the teaching of diplopia to patients with NRC, there are several goals at which we must aim: (1) Diplopia must be spontaneous with steady fixation, not quick alternation. (2) Diplopia must be spontaneous with either eye fixing. (3) Diplopia must be spontaneous at any angle of divergence, especially at the usual angle of squint. (4) Diplopia must be spontaneous with fixation on any object, not just on a fixation light.

For whom: Patients with normal retinal correspondence
To teach: Diplopia with steady fixation

Diplopia must be spontaneous with steady fixation, not rapid alternation. This is difficult to teach to small children. When I am unable to get them to look steadily at one light while telling whether they can see the other I find it very helpful to send them home with what I call the "peripheral vision" exercise. The patient practices looking directly at his mother's face or at some object which she holds up in front of her nose while he discusses other objects which are visible to the side. His mother has objects of bright, solid colors (scarves, pieces of construction paper, blocks, or whatever is available) which she holds up beside her face for color recognition while the patient continues to look steadily at her. "No fair peeking; look right at mother's nose." This exercise can also be carried on at the dining room table where, while watching his father, the child can see out of the corner of his eye when his mother takes a drink of coffee, etc. On the return visit the patient should be aware of the fact that when he is looking at the red light it may be possible to see the white light without turning his eyes to find it. He is then ready for actual diplopia training.

As many reds as are necessary are used over the fixing eye, and the patient fixes the red light steadily, seeing the white light "all fuzzy and blurry out of the corner" of his eye. (If he doesn't know what blurry means, I hold a high plus lens before the fixing eye to show him.) The darker the room and the brighter the fixation light, the easier it is to obtain diplopia. The reds are gradually reduced in number while the patient maintains diplopia. When the second (white) light is lost, one red is again added, the white light found, and the reds again reduced. Since diplopia

is a function of the brain, the patient must be thinking about the position of the white light at all times. My line of patter runs something like this: "Now while you're looking right at that red light, can you see the white light? Keep looking right at that red light. Can you see the white? It's all blurry, isn't it? Which side is the white light on? That's right. Now see where it is? When I take one of these reds away, see if you can still see the white light. Did it go away? Remember where it was and pretend you can still see it. There, see it now? When I take this last red away, keep looking at the light on this side (as I tap an arm or a cheek) while it changes to white. Do you still see two lights? No, don't look around—keep looking at *this* one." I try as much as possible to avoid using the word "think," since I am always being confronted with the fact that small children don't know the meaning of the word. Some of them even have the audacity to ask me, and I haven't yet found a satisfactory answer.

Before I send a patient home with the peripheral vision exercise, there are several things which I do. One is to check his answers by covering his nonfixing eye and asking whether he still sees one white and one red light. Another is, when I am explaining it to the child's mother, to show her with a prism before her eye just what the patient should see. I am very careful to tell her that she and the other members of the family should not worry because they can't see double—that diplopia is present only when the eyes are not straight. And since her eyes are straight, she cannot see double except with a prism before one eye, which is like making believe that her eyes are crossed.

For whom: Patients with normal retinal correspondence

To teach: Diplopia with either eye fixing

Have you ever had a patient who seemed to have excellent diplopia; he could see double the minute you asked him to; he had seen double at least 100 objects a day at home just as you had asked him to do; and still his mother reports that his eye turns when he seems unaware of it? I have had many such patients. It took quite a while for me to realize that diplopia is easy when fixing with the usually nonfixing eye, and that is what these patients were doing. They were looking with the usually suppressing eye when they wanted to see double, and looking with the usually fixing eye when they wanted to suppress. Consequently, a most important factor in diplopia training is that it must be present when the usually fixing eye is fixing. So if the patient has a right esotropia (left eye straight), we work on diplopia while he fixes with the left eye and keeps the fuzzy image on the right side. I have the patient with a monocular squint wear something on the hand which is on the same side as the fixing eye. For instance, in a right esotropia, the patient wears a ring or bracelet on the left hand reminding him that he is to look at the image with his left eye. If the patient is an alternator, either naturally or because of occlusion, diplopia must be worked on with each eye fixing; sometimes keeping the fuzzy image on the left, sometimes keeping it on the right.

For whom: Patients with normal retinal correspondence

To teach: Diplopia with any amount of squint

Diplopia must be spontaneous with any amount of squint. Surely you have seen, as I have, patients with a small area of suppression who increase or decrease their angle of squint in order to get diplopia. I see this action particularly in persons with exotropia who converge slightly to get double vision

easily. Still they can return their eyes to their old angle of squint and suppress. They must be taught to relax to their usual angle if they overconverge, or to "look hard" if they relax their angle to obtain diplopia. I am also careful to show the child's mother how to watch the light reflex in the nonfixing eye to catch any change in the angle of squint. Persons with accommodative squint are often difficult subjects for training in diplopia. You think that they are doing beautifully—getting diplopia spontaneously at their usual angle of squint, and seeing clearly with their eyes straight. However, I've found that they have to be watched very carefully, because some of them (the bright ones), instead of really straightening their eyes to fuse, bring them almost straight and then suppress. A patient who does this can bar read because light rays from the bar fall on temporal areas in each eye, areas which a person with esotropia has never had to suppress. He can suppress the nasal area of the nonfixing eye on which the print is falling, but still he can not suppress the temporal areas on which the bar is falling. He must be taught to see double even when his eyes are only slightly crossed, as well as when they are crossed the usual amount.

For whom: Patients with normal retinal correspondence
To teach: Diplopia on all objects

It is not enough to be able to see two lights at distance and at near in the office and at home. That diplopia must be transferred to objects; otherwise we have the same situation discussed previously— the patient sees double when he wishes, and suppresses at other times. This difficulty is overcome gradually. I usually have the patient's mother put the light on her nose. When the patient can see two lights, we try to convince him that his mother must have two noses—one behind each light. Sometimes a sticker on her nose helps. If necessary, the red kodaloid is again used until the patient can see two noses; the red is then removed. If he continues to see two noses, the light is removed. The patient holds the image of two noses, using the light when necessary, but remembering that the goal is to see two noses without the light. If two noses are seen there must be two mouths, then four eyes, then finally two faces. If the squint is present only at distance, the light is placed near some object (under a picture, in front of a glass or cup) and the diplopia is transferred from the light to that object. When the patient can get diplopia on objects without the use of the light, he practices on at least 100 objects a day. He makes a "diplopia" tour of the house, seeing two of everything. He practices seeing two of each of his friends, two bus drivers, two trees, two swings, etc. At this point we begin to try to straighten the eyes. In constant squint, occlusion is used until the patient can learn to straighten his eyes himself, or until they are corrected surgically.

For whom: Patients with normal retinal correspondence who are unable to achieve diplopia with red glass
Exercise: Use of prisms to elicit diplopia

If, for some reason, the patient cannot appreciate diplopia while using several layers of red glass and does not have a blindspot mechanism, I use a prism. This can be done in one of several ways, depending upon the type of squint and the area of suppression.

1. If a patient has a constant tropia with a particularly stubborn small area of suppression and has trouble producing regular red-glass diplopia, the use of a small moving prism may be helpful. I use a prism of 5△ or so, either base in or base out, over the nonfixing

eye. This is used off and on, off and on, making the image appear to move back and forth. Since a moving object is less easily suppressed than one which is still, the patient is likely to see this image as it moves. He must then concentrate on its two positions. I often ask a small patient to walk to the other end of the room and show me where the two positions of the light were. By remembering where the light was, and by imagining he can see it there, often he really can see it. If not, the exercise is repeated. If necessary, a red glass is used in conjunction with the prism. After diplopia can be held with a light, this same exercise can then be repeated on small objects.

2. If the squint is purely horizontal and variable, the suppression area is likely to include most (or all) of the rods and cones between the macula and the part of the retina where the image falls when the squint is the greatest. In a convergent squint this is, of course, the nasal retina; in a divergent squint, the temporal. If such patients do not get diplopia with a red glass, I try one of two things, both with a vertical prism. The vertical prism, either base up or base down before the nonfixing eye, throws the image of the fixation light above or below the horizontal suppression area.

a. This vertical prism may be used off and on, off and on, making the image appear to jump up and down. If done rapidly, the image on the usually suppressing area is seen. The patient is told to keep fixation on the image while it jumps, and the movement is slowed down until eventually all movement is stopped and the prism is removed entirely.

b. In the other procedure, the vertical prism may be held quietly before the nonfixing eye and slowly rotated so that the image falls on the usually suppressing portion of the retina. Since a small vertical prism is usually sufficient, I rotate the prism sometimes to base in and sometimes to base out. Usually suppression will again occur just before the prism has reached the horizontal position, in which case we try again. When suppression does not recur during this exercise, usually the patient is ready to attempt diplopia with a red glass. The latter is practiced at distance and near, while accommodating and while not accommodating, making the squint larger and smaller to eliminate suppression on all peripheral areas of the retina.

For whom: Patients with normal retinal correspondence
What: Postoperative instructions given preoperatively

When a child with squint and NRC is going to have surgical treatment, I like to save the last preoperative visit until after the child knows of the impending operation. I explain to him and to his mother that it will be several days after the operation before his bandages are removed, and that as soon as they are, his job will begin. Since I will not see him until the tenth postoperative day, he will have a good week to work hard by himself. He is to see everything double. Of course, he may do this spontaneously. Then it will be his job to put the double images together. We practice this preoperatively on the troposcope. I set the machine at slightly less or slightly more than his angle of squint, and we pretend that he is seeing double after his bandages have been removed. Then he tries to put the two images together. Actually, if diplopia is spontaneous preoperatively and the patient is told that after surgery he may see double and it will be his job to put the two images together, the results are often remarkably good.

For whom: Patients with intermittent squint
To teach: Diplopia before straightening

In regard to diplopia training in cases of intermittent squint, I find that in accommodative squint and intermittent exotropia, the training is much faster if diplopia is spontaneous *before* the patient is consciously taught to straighten his eyes on command. If the straightening is taught first, I find that often the patient cannot then squint voluntarily – only unconsciously, and diplopia training is then nearly impossible. I always explain several times to the patient that "two means crossed or crooked" and "one means straight." With young children we make a question-and-answer game of that: "Are your eyes straight or crossed when you see two?" "What should you see when your eyes are straight?" In accommodative squint it is important that the patient *not* learn that "blurry means straight" and "clear means crossed." It is very difficult to teach dissociation of accommodation and convergence to those who have learned that "blurry means straight." They don't want to clear the image without glasses, since "clear means crossed."

For whom: Patients with intermittent tropia whose eyes stay straight under observation
Exercise: Use of prism to simulate squint

If the patient has an intermittent tropia and can't turn his eye on command to practice diplopia, a prism which throws the image toward the usually suppressing part of the retina is used. In other words, for a patient with intermittent esotropia of 20△ who squints only when unaware of his eyes, I try a prism of 20△, held base in, over the usually suppressing eye. The exercise is then continued with a red glass just as though no prism were being used. (A word of caution is necessary at this point. The eye under the prism should be watched. The idea of the prism is to simulate the usual angle of squint. However, if the 20△ prism, in breaking up fusion, creates by its own 20△ and the patient's 20△ a 40△ squint, this prism should not be used. Possibly a 5△ prism will break up fusion enough to produce an image much closer to the usual area of suppression by giving the patient very nearly his usual amount of squint.)

For whom: Patients with intermittent squint with good diplopia
Exercise: Race between mother and child

When a patient with intermittent squint has learned to straighten his eyes voluntarily and has good diplopia which is almost spontaneous, we begin the race between mother and child. The child, when in the presence of his parents, announces aloud every time he sees double without trying, that is, spontaneously. I explain that this is so important that the child may interrupt any conversation to say "two." His mother keeps a record of how many times the child says "two." She also keeps a record of the number of times she noted a squint when the child did not announce his double vision. The idea is for the child never to let his mother see a squint without his having first said "two." The rapid decrease in the number of times the mother notes a squint is amazing.

For whom: Patients with squint who have normal retinal correspondence with some fusion
Exercise: Physiological diplopia

Physiological diplopia is given, of course, only to patients with normal fusion at some distance – this may be

from two inches in front of the eyes to 20 feet. Most exercises for physiological diplopia utilize only one type of diplopia, i.e., crossed-homonymous diplopia. If the fixation point is a near object, the distant object will appear as homonymous diplopia; if the distant object is the fixation point, the near object will appear as crossed diplopia. For patients with intermittent esotropia, the near object is more valuable as a fixation point than the distant one; since most of them can already get spontaneous crossed diplopia if the temporal areas of the retina are stimulated, they have no need to suppress this area. I give these patients an exercise which is the opposite of framing; that is, using the finger as a fixation point, they try to put the finger exactly in the middle of the double image that they see across the room. Patients with intermittent exotropia, on the other hand, are given framing exercises, since with fixation on an object across the room, light rays from a finger held directly in front of the nose fall on temporal portions of both retinas, the usually suppressed areas. If physiological diplopia is very difficult, we begin by using a light as the object of diplopia – across the room for patients with esotropia or held in the hand for those with exotropia. If necessary, just as in regular diplopia exercises, a red glass is used.

For whom: Patients with normal retinal correspondence and fusion within 6 feet
Exercise: Physiological diplopia with string

One exercise for physiological diplopia utilizes both crossed and homonymous diplopia and can be used for patients with constant esotropia, provided that they have NRC and either no hypertropia or a hypertropia corrected by prisms (at least during the exercise periods). A string, 5 or 6 feet long, is attached at one end to a stationary object while the other end is held on the tip of the patient's nose. If a pencil is held against the string at the point where his visual axes cross, fusion will occur. At the same time that part of the string which is beyond the pencil will appear double, as homonymous diplopia of an increasing amount. Likewise, the part of the string which is closer than the pencil will appear double, as crossed diplopia. Thus, when fusion takes place the string appears to be two strings which cross each other at the point of fixation. In constant esotropias this point of fixation will be at any place where the visual axes cross – two inches from the nose, four inches from the nose, etc. The patient is told to put his finger on the place where the strings appear to cross. Then at that point I hold a pencil. As he sees one pencil and two strings *both in front of and beyond* the pencil, I gradually recess the pencil as long as he can keep fusion and both crossed and homonymous physiological diplopia. When he loses fusion (sees two pencils or suppresses one string) I advance the pencil toward him slightly until he regains fusion. If necessary for diplopia a red glass is used to obtain the two strings. It serves also to check the patient's answers (the white string appears to be coming from the eye covered with the red glass and a red string from the other eye – a fact which the patient doesn't guess if he doesn't actually see it). The object is to be able to fuse anywhere along that string keeping at all times both crossed and homonymous diplopia at all points other than the fusion point.

For whom: Patients with anomalous retinal correspondence
Exercise: Diplopia

Is there any diplopia exercise that can be given to a patient with ARC?

Well, that depends upon the depth of the ARC and on the age, intelligence, and cooperation of the patient. Quite a few years ago Julia Lancaster told us of a diplopia exercise which she gives to patients with ARC. At the time I threw up my hands in utter astonishment and refused to try it, saying that it could have no value since the technician could not be watching the patient's eyes. I was new in orthoptics and too insecure in my thinking to try anything so far removed from what I had been taught. And maybe that is just as well for someone new in orthoptics. But I am glad I heard of it, because now I wouldn't be without that exercise. The principle of the exercise is to change a paradoxical diplopia into a normal one. This can be done in one of two ways:

1. A prism which nearly corrects the esotropia usually produces a crossed diplopia in a patient with convergent ARC. With thought and imagination he tries to get those images very close together – in a homonymous position. I explain to the patient that this is not done with his eyes – he must not turn one eye in further – but it is done with his brain. That is, he must pretend that he can see the lights close together; think about it (these patients are old enough to know what "think" means); concentrate on it. If he can get a normal projection, even momentarily, in the office, I give this as a home exercise. The working distance at home must be taken into consideration, since the patient's angle of squint may vary at different distances. That is, he may have an esotropia of 20△ at 20 feet and an esotropia of 30△ at 10 feet. So if the working distance at home is to be 10 feet, the patient's angle of squint should be measured at that distance and the correct prism prescribed.

2. When he can make the images touch with the prism, we progress to a normal diplopia without prisms. Again the working distance at home must be considered. Say the working distance is to be 3 feet (1 meter). A normal separation at that distance would be 1 cm. for each prism diopter (30 cm. for a 30△ squint). That is how far apart the patient should see the two lights. If they are considerably closer together, the patient is using his ARC, and he must concentrate on the place at which his mother shows him the light should be.

A couple of things should be kept in mind with this exercise: (1) In the office the patient's eyes should be watched carefully, for if he tends to turn one eye in more than usual when trying to get the "correct" separation of the lights, he should not do the exercise at home. (2) The patient must be old enough to understand what he is doing and why. (3) The patient's answers must be reliable.

For whom: Patients with anomalous correspondence
Exercise: Monocular diplopia on major amblyoscope

How can we use monocular diplopia in treatment? One way is the Walraven technique, which is given in full in Mary Kramer's book.[1]

I may be a bit old fashioned, for I use the oscillation technique. However, my line of patter is entirely different than it used to be. I still begin with large second-grade targets, because I feel that peripheral fusion is easier than macular or foveal fusion. I like particularly the silhouette targets which come with the troposcope or which I make myself by gluing stickers such as large stars, circles, and squares, on paper. Instead of the constant shaking and "Tell me when you see one house" technique, my patter runs something like this: "See the house on this side?" (I touch the left hand.) "It has a moon, hasn't it? Now keep watching that

house and tell me when it gets a star, too." The normal projection of the star (above the house with the moon) may be only momentary at first, but gradually it remains for longer periods of time. The abnormal image is often suppressed unless attention is called to it. If we have been talking about the normal image and I ask if the other has been there all this time, the answer is usually "I haven't even noticed." And what is that if not suppression?

For whom: Patients with anomalous retinal correspondence

Exercise: Monocular diplopia off major amblyoscope

Just as we used monocular diplopia on the machine, we can also in some cases use it off the machine. I use a red glass and prisms which nearly equalize the squint. A patient with convergent squint and ARC will then have crossed diplopia, with the images fairly far apart. However, if he concentrates on it, he can often find another red light close to the white light. This, of course, would be the normally projected image. The biggest obstacle in obtaining monocular diplopia is a mental block which says, in effect, "It's impossible to see two things with one eye." I usually explain to the patient that those lights should be close to each other, and really are close to each other, but his brain just doesn't want to see them that way. I explain that there is nothing wrong with his eyes that makes him see the light way over there; it is his brain which does it. We talk about imagining the light is in the right place, and thinking about it. He soon gets it there – at first momentarily only, then for longer and longer periods.

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1. Kramer, Mary: Clinical Orthoptics, ed. 2, St. Louis, C. V. Mosby Co., 1949, pp. 384-386.

THE PRACTICAL SIGNIFICANCE OF THE SUBJECTIVE ANGLE

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ALL workers in the field of ocular motility realize how difficult it is to measure the angle of deviation in children, in whom squint is found most frequently. It is difficult because of the variability of the cooperation of these children and because of the variation in the angle of squint. If we are to treat these patients we must have some idea of the actual amount of deviation that is present. This means that it is necessary to know what the usual angle of deviation is when the child is at play or work and not conscious of his eyes or of being observed. I like to think of this as the basic angle of squint, or the angle at which the eyes are positioned most of the time. Now, in many cases it is easy to determine this basic angle with the prism cover test and by measuring the objective angle on the troposcope. However, these tests, by their very nature, break up fusion and because of this may give an erroneous result. For example, as you know, in some patients the eyes can actually be straight, but when fusion is disrupted, a large angle of squint will be measured.

Because of this variability in the results of objective measurements there recently has been a tendency among ophthalmologists to shy away from quantitative measurements altogether. But it is in just these very variable cases that a method of mensuration would be extremely valuable. In these cases orthoptic treatment, if properly carried out, gives the best hope for a functional cure, since contracture of muscle and fascia and abnormal correspondence have not been consolidated.

Von Graefe was one of the first to use a subjective method for the measurement of the deviation in cases of recent muscular paralysis. His technique was to chart the diplopia fields, as obtained with a red glass over one eye and a candle as the target. He found that the test was also of value in indicating what muscle was at fault and the result of treatment. The importance of subjective testing has been confirmed, and it is in routine use in most eye clinics for cases of muscle paralysis. The more refined methods in use today are the Hess screen test and the Lancaster red-green test.

Since the measurement of the subjective angle of squint has been demonstrated to be so accurate in cases of recent muscular paralysis, would it not be of value in cases of concomitant squint with normal correspondences? I have found that the subjective angle of deviation, as measured on the troposcope, could give information about the basic angle of deviation in some young children that was not obtainable by other means. This was because of a characteristic reaction that occurred when the children were placed in front of the instrument. Some, at first, went all to pieces, and no reliable answer could be obtained. However, many children, after a while, found this a fascinating game and became so absorbed in putting the lion in the cage that they forgot all about their eyes. When this happened it was possible to determine the basic angle of deviation.

From the Motility Clinic of the Illinois Eye and Ear Infirmary, University of Illinois College of Medicine.
Presented at the Fifteenth Annual Meeting of the American Association of Orthoptic Technicians, Oct. 14-16, 1956, Chicago.

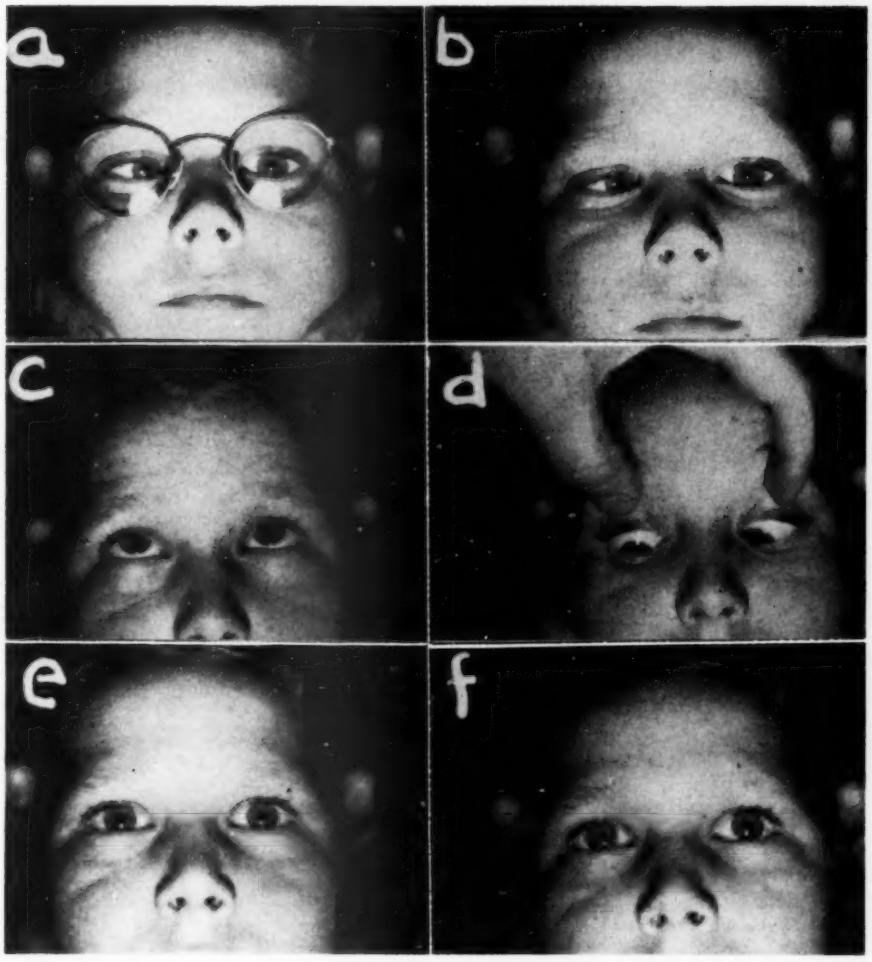

FIG. 1 – Case 1. (a and b) 20 degrees of right esotropia with and without glasses; (c) about 5 degrees of esotropia on looking up; (d) 20 degrees left esotropia on looking down; (e) eyes straight for distance; (f) eyes straight for near.

In this paper I will attempt to show, by means of case histories, how the subjective angle, as elicited on the troposcope in cases of concomitant squint with normal correspondence, has been of practical value in diagnosis and treatment.

CASE REPORTS

Case 1. A 6.5-year-old boy came to the Infirmary in September 1946 with a history of having had crossed eyes for the past six months following a severe attack of measles. Results of refraction with atropine cycloplegia were O.D., +1.00 sph., visual acuity 20/20; O.S., +.75 sph., visual acuity 20/20. There was 20 degrees of alternating esotropia under atropine. The boy was sent to the orthoptic clinic for evaluation and treatment. It was decided to give him a course of fusion training, since the squint was of such recent onset. Examination revealed 20 degrees of esotropia was present with and without glasses. Cover measurements were 50△ esotropia for distance; 50△ esotropia for near. Second-grade fusion was found at the objective angle of 54△ base out. This patient had normal retinal correspondence and was able to diverge from 54△ base out to 40△ base out.

After a course of fusion exercises twice a week for one year, the findings were as follows (fig. 1, a, b, c and d): There was still 20 degrees of esotropia with and without glasses.

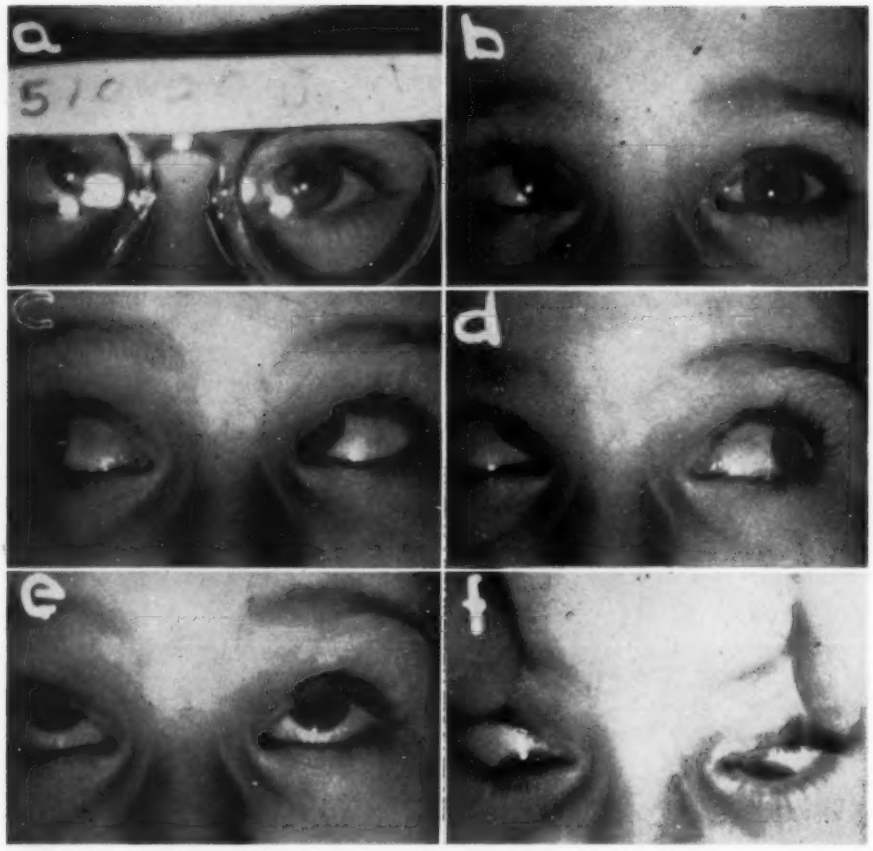

FIG. 2 – Case 2, April 1950. (a) 25 degrees of right esotropia and 10 degrees of right hypertropia with glasses; (b) 30 degrees of right esotropia and 10 degrees of right hypertropia without glasses; (c and d) marked bilateral elevation in adduction; (e) about 5 degrees left esotropia on looking up; (f) 25 degrees of esotropia on looking down.

With glasses the cover measurements were 43△ esotropia for distance, 45△ esotropia for near; without glasses the cover measurements were 40△ esotropia for distance, 44△ esotropia for near. There was second-grade fusion at the objective angle of 44△ base out with amplitude to 26△ base out on the synoptophore. A note on the orthoptic record stated that the eyes could be straight on casual gaze at times. The boy was referred to me for consideration of surgical intervention or suggestions for further treatment.

The day I saw him, his eyes were straight when he walked into the clinic (fig. 1, e and f). Before I could examine him, he was placed on the rotoscope and given a diverging treatment. When I saw him again after the exercise, his eyes were crossed 20 degrees, and they remained crossed while I examined him. This was very confusing! He had come in with apparently straight eyes, and after a treatment, he had left the clinic with crossed eyes.

I decided that the exercises might be making the child's eyes worse and suggested that all treatments be stopped. When he returned to the clinic two months later, his eyes were straight. The straight position of his eyes in casual gaze was a clue that perhaps this was his basic position. To test this, he was examined on the synoptophore with second-grade targets set at 0, and he was able to fuse easily. He could not fuse third-grade targets. But when the attempt was made to get the objective angle, he promptly developed a large esotropia. Because of the small basic angle of squint, it was decided to follow the progress of this patient without resorting to surgical treatment.

When last examined, in June 1954, the eyes were straight in all positions; the only abnormality was an esophoria of 3△. He fused

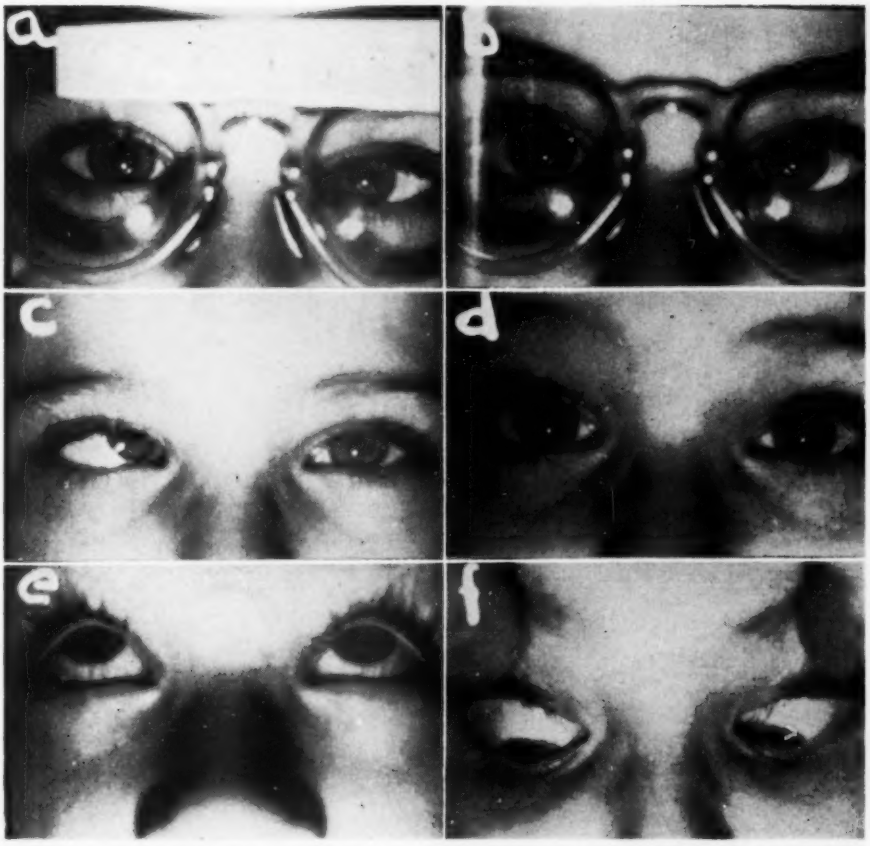

FIG. 3 – Case 2, May 1950. (a) 15 to 20 degrees of left esotropia with glasses for near; (b) 10 to 15 degrees of left esotropia with glasses for distance; (c) 40 degrees of right esotropia; (d) about 5 degrees of right esotropia; (e) eyes straight on looking up; (f) 15 to 20 degrees of left esotropia on looking down.

third-grade targets with good amplitude. In this case the subjective angle findings were of importance in that they prevented unnecessary surgical intervention.

Case 2. A 5-year-old girl, came to the Infirmary on April 5, 1950, with a history of the left eye having started turning in at the age of 2.5 years, intermittently at first, then constantly. She was wearing her full atropine correction, which was O.D., +4.50 sph. +.50 cyl., axis 90, visual acuity 20/30; O.S., +3.50 sph. +1.25 cyl., axis 115, visual acuity 20/30. She had been treated for a left amblyopia and had had a course of orthoptic treatments. She was referred for surgical operation by her attending ophthalmologist.

Examination revealed 25 degrees of esotropia with glasses and 30 degrees without (fig. 2). A large amount of hypertropia was also present. Marked bilateral elevation in adduction was seen. There was 5 degrees of esotropia on upward gaze and 25 degrees on downward gaze. The NPC was 30 mm. Certainly with these findings an operation was indicated. The child was sent to the orthoptic department for preoperative prism cover measurements and troposcope findings.

In May 1950 with glasses the cover measurements were 24△ esotropia, 4△ right hypertropia for distance; 34△ esotropia, 2△ right hypertropia for near. Without glasses the measurements were 56△ esotropia, 18△ right hypertropia for distance; 54△ esotropia, 16△ right hypertropia for near. The objective and subjective angle was found at 36△ base out, 10△ right hypertropia, with first-grade targets. However, a note was made that second-grade fusion was found at zero.

When I noted the findings of second-grade fusion at zero, two possibilities came to mind: (1) this could be a case of harmonious anomalous retinal correspondence, which is rare;

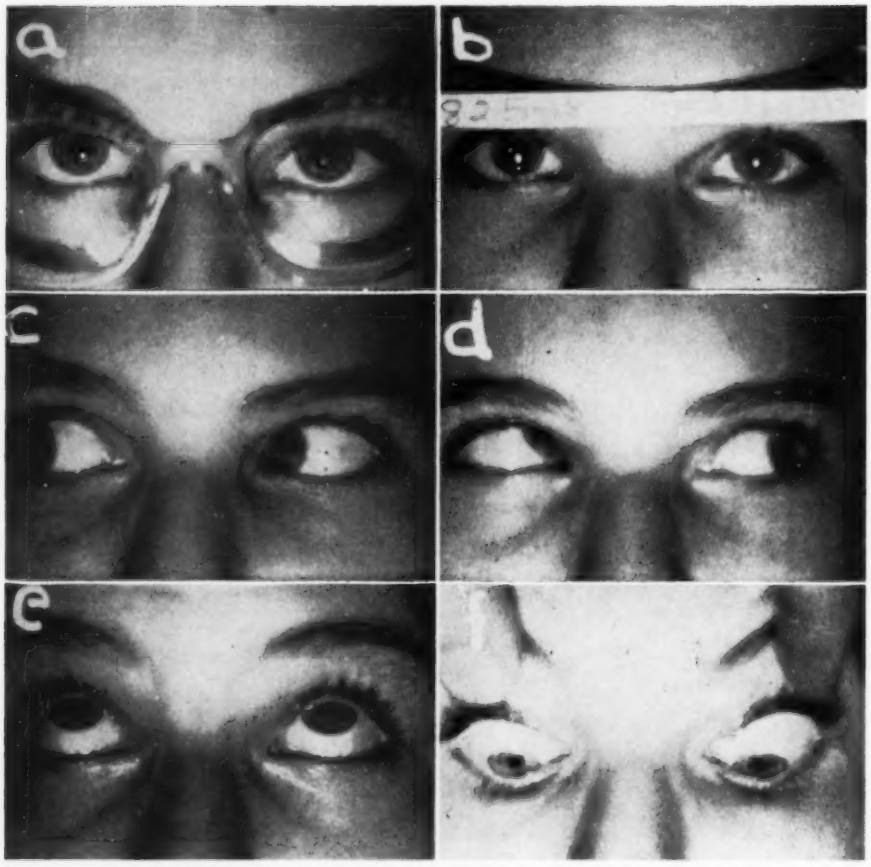

FIG. 4 – Case 2, June 1956. (a and b) Eyes straight with and without glasses; (c) elevation of the left eye in adduction much less; (d) marked elevation of the right eye in adduction; (e) eyes straight on looking up; (f) eyes straight on looking down.

(2) the basic angle of squint could be at zero, indicating that the child had normal retinal correspondence. It was necessary to re-examine this patient very carefully.

After careful study it was possible to elicit a much smaller angle of squint than was found at the first examination one month earlier; in addition, her eyes could straighten out until they appeared almost parallel (fig. 3). On the troposcope, when the patient fused second-grade targets at zero, examination of the corneal reflexes showed the eyes to be straight. In view of these findings there was a definite possibility that the eyes could be straight a good deal of the time. Consideration of surgery was delayed; the patient was placed under observation.

Examination in June 1956, 6 years later, revealed that the eyes could be straight with and without glasses (fig. 4). The elevation in adduction of the left eye was lessened. The eyes were straight on looking up and straight on looking down, and the NPC was 110 mm. With glasses the cover measurements were esophoria to 7△ esotropia, 2△ right hypertropia for distance; 22△ esotropia, 4△ right hypertropia for near. Without glasses the measurements were 54△ esotropia, 10△ right hypertropia for distance; 58△ esotropia, 14△ right hypertropia for near. There was second-grade fusion at zero.

In contrast with case 1, it was not immediately observed at the clinic that this patient's eyes were straight on casual gaze. This was because we really did not study her carefully enough, since she had been referred to us for surgical treatment by her own ophthalmologist, who had been following her case for two years. The first clue to the situation was the finding of straight eyes when the subjective angle was determined. When this was noted, it was easy to observe that her eyes were

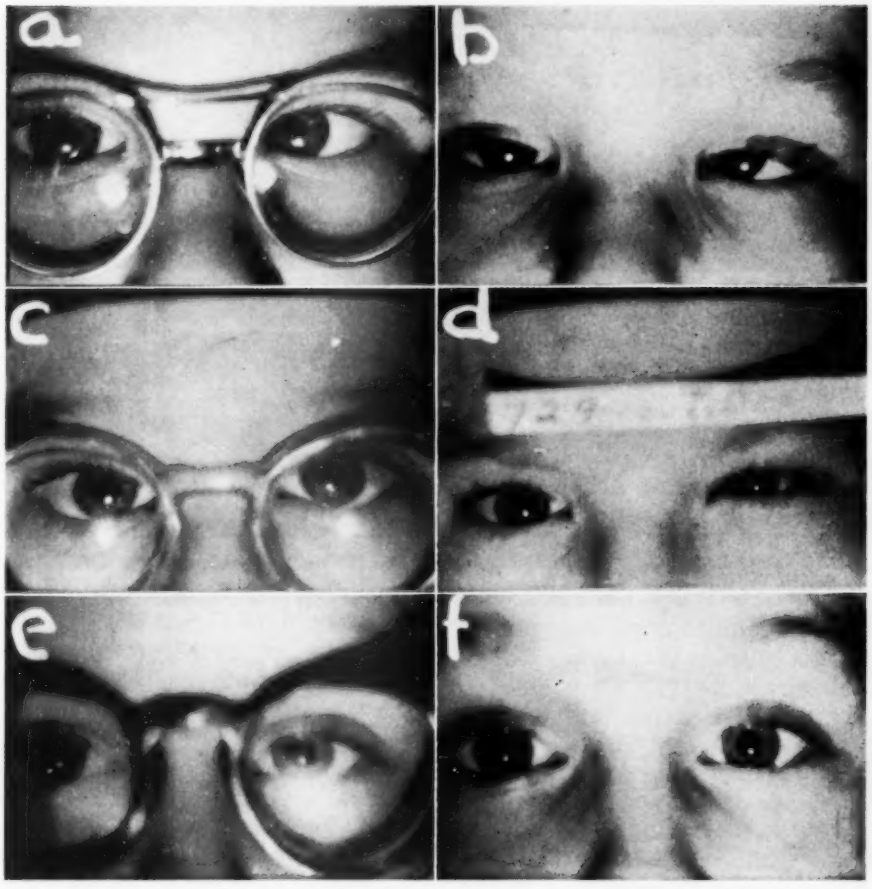

FIG. 5 – Case 3. (a and b) August 1949, 20 degrees left esotropia with and without glasses; (c and d) July 1954, eyes straight with glasses and 15 degrees of left esotropia without glasses; (e and f) December 1955, eyes straight with and without glasses.

straight on casual gaze on her subsequent visits to the clinic. She has gradually improved so that the eyes are straight all of the time, even with cover measurements.

Another important finding was the change in the amount of hypertropia. With glasses, for distance, only a right hypertropia of 2△ was found, while without glasses, a right hypertropia of 10△ was seen. On the troposcope, no hypertropia was found when the patient fused. The finding of fusion at zero demonstrated that the hypertropia, by disappearing with fusion, was secondary to the dissociation of the eyes by the esotropia and was not due primarily to a paretic vertical muscle. Again, as in case 1, a knowledge of the subjective angle prevented unnecessary surgical intervention.

Case 3. A 4-year-old boy, came to the Infirmary in August 1949, with a history of the eyes having turned in since birth. Results of refraction with atropine cycloplegia were O.D., +6.00 sph., +1.00 cyl., axis 90, visual acuity 20/30; O.S., +6.50 sph., +1.25 cyl., axis 90, visual acuity 20/30. Examination revealed 20 degrees of left esotropia with and without glasses (fig. 5, a and b).

Surgical correction was decided upon because of the large deviation, present since birth, upon which glasses apparently had little effect. The boy was sent to the orthoptist for a preoperative workup. To our surprise the prism cover measurements, with glasses, were only 10△ esotropia for distance; 18△ esotropia for near. Without glasses the measurements were 25△ esotropia for distance; 35△ esotropia for near. This demonstrated that he had a much smaller squint than had been evidenced at the clinic and that an accommodative component was present. On the troposcope with

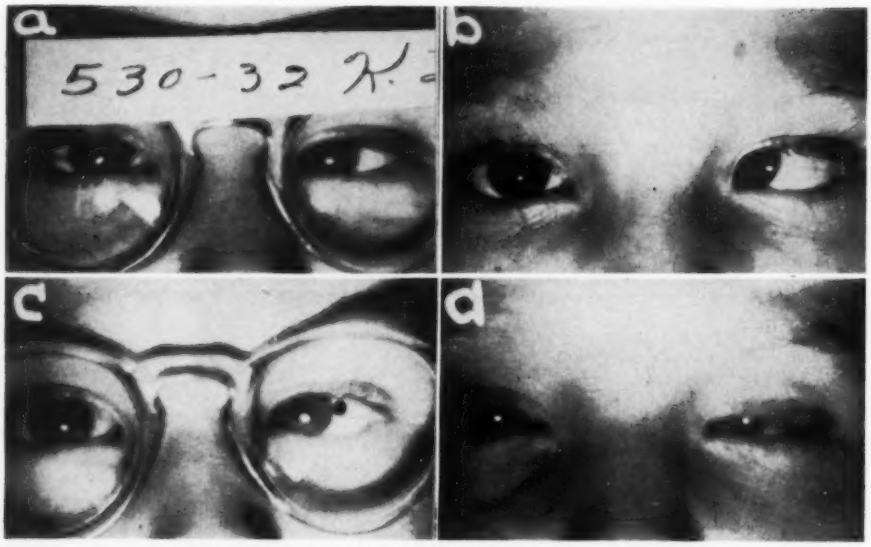

FIG. 6 – Case 4. (a and b) August 1950, preoperatively, 20 degrees of left esotropia with glasses; 30 degrees of left esotropia without glasses. (c and d) September 1950, two months postoperatively, 15 degrees of left esotropia with glasses; 20 degrees of left esotropia without glasses.

FIG. 7 – Case 4. (a) June 1956, 15 degrees of left exotropia with glasses. (b and c) August 1956, eyes straight with glasses; 20 degrees of left esotropia without glasses.

glasses there was second-grade fusion at the objective angle of 10△ esotropia, 2△ right hypertropia. This confirmed what was found with the cover measurements – that a small basic angle of deviation of about 5 degrees was present with glasses. The operation was cancelled, and the child was placed under observation. The eyes remained crossed a good deal of the time during the ensuing years, but they did improve slowly. Five years later, in July 1954, the eyes could be straight with glasses, while without glasses there was still 15 degrees of esotropia (fig. 5, c and d). In December 1955, the eyes could be straight with or without glasses (fig. 5, e and f).

The orthoptist was to be congratulated because it was due to her findings that this child was not operated on, even though on many occasions his eyes were so crossed that we doubted the wisdom of our decision. The cover measurements with glasses, were now orthophoria for distance, 5△ esophoria for near. Without glasses the measurements were 9△ esotropia for distance, 20△ esotropia for near. The NPC was 50 mm.

This appeared to be a good result. But here again the importance of the subjective angle was demonstrated. With glasses, second-grade fusion was found at 12△ base in. This indicated that, in spite of the straight-appearing eyes and the orthophoria with cover measurements, actually an exotropia was developing. A reduction in the strength of the lenses was urgently needed to prevent the development of a permanent exotropia.

Case 4. A 5-year-old girl, came to the Infirmary in April 1950, with a history of crossed eyes since she was 20 months of age. Results of refraction with atropine cycloplegia were O.D., +4.00 sph., +2.75 cyl., axis 80, visual acuity 20/30; O.S., +3.50 sph., +3.00 cyl., axis 80, visual acuity 20/30. Examination revealed 20 degrees of esotropia with glasses and 30 without glasses (fig. 6, a and b). Prism

cover measurements with glasses were 25△ esotropia for distance; 35△ esotropia for near. Without glasses the measurements were 50△ esotropia for distance, 50△ esotropia for near. On August 20, 1950, a 5-mm. recession of the right medial rectus and a 10-mm. resection of the right lateral rectus muscle were performed. Examination, two months after the operation, revealed 15 degrees of esotropia with glasses and 20 without glasses (fig. 6, c and d). This was a marked undercorrection, and she was referred for an orthoptic workup with the possibility of reoperation in mind. Fortunately the child was old enough to be examined with some degree of accuracy. It was found that the cover measurements with glasses were 7△ exotropia, 4△ right hypertropia for distance; 5△ esotropia, 2△ right hypertropia for near. Without glasses the measurements were 16△ esotropia, 2△ right hypertropia for distance; 23△ esotropia, 2△ right hypertropia for near. Second-grade fusion was found at 4△ base in.

These findings were exciting! Here was a child who always had had a large esotropia with the Hirschberg test when seen in the clinic, yet, when examined under different conditions by the orthoptist, she had a definite exotropia for distance, both objectively and subjectively. Should the measurements be disregarded, as some ophthalmologists would do, for not being too accurate in children anyhow, and should her eyes be surgically treated for the amount of squint observed in the clinic? Because our experience (in cases 1, 2, and 3) indicated that measurements were of significance when the patient was followed over many years, it was decided not to reoperate and, with keen interest, the patient was studied periodically in the motility clinic.

Over the years a definite esotropia was observed at all times, both for distance and for near, even when glasses were worn, and many times her eyes looked pretty badly crossed. Both the parents and I became discouraged at times. When the child was examined in June 1956, after a six-month absence from the clinic, a radical change in the squint had taken place. Now, for the first time, an exotropia of 15 degrees was observed while she was wearing glasses (fig. 7, a). She was promptly examined on the troposcope, and second-grade fusion was obtained at 4△ base in with glasses and at 4△ base out without glasses. With these findings of exotropia with glasses, an immediate reduction in her lenses was indicated, and each lens was weakened by 2.00 diopters.

Two months later, re-examination showed that the exotropia was no longer present and that the eyes were straight with glasses (fig. 7, b and c). Without glasses, 20 degrees of esotropia was present. Cover measurements were orthophoria for distance and for near with glasses. Without glasses the measurements were 42△ esotropia for distance; 40△ esotropia for near. Second-grade fusion was found at 4△ base out with glasses.

Now a new problem presented itself. Were the large squint without glasses and the troposcope findings of fusion at 4△ base out both warning signs that the lenses were reduced too much? The answer to this question was not of theoretical interest alone, since I have had the experience of reducing a patient's lenses too much and having him develop an esotropia that remained even when the stronger lenses were replaced. On the other hand, patients whose lenses were not reduced soon enough have developed an exotropia which did not disappear when the lenses were removed.

It would seem that in these cases in which a change of lenses can produce a large effect on the position of the eyes, changing it from straight to esotropia or exotropia, an accurate method of determining the strength of lenses to be prescribed would be extremely useful. I have found that determination of the subjective angle has been of value in ascertaining what the proper correction should be. For example, in the case above, the subjective angle with glasses will be measured at two-week intervals. If any increase in the base-out angle is found, stronger glasses will be prompt-

ly ordered. Our purpose will be to keep the subjective angle at zero.

In the next case, the patient developed a postoperative exotropia because the subjective angle was not taken into consideration before and after the operation.

Case 5. A 10-year-old boy came to the Infirmary in August 1947, with a history of his eyes having been crossed since he was 18 months of age. Results of refraction with atropine cycloplegia were O.D., +3.00 sph., visual acuity 20/20; O.S., +3.00 sph., visual acuity 20/20. About 25 degrees of esotropia was present with glasses and around 30 without glasses (fig. 8, a and b). Cover measurements with glasses were 48△ esotropia for distance; 55△ esotropia for near. Without glasses they were 55△ esotropia for distance; 60△ esotropia for near.

On October 1, 1947, a bilateral 10-mm. resection of the lateral rectus muscles was done. Examination, two months after surgery, revealed about 10 degrees of esotropia with glasses and about 20 degrees without glasses (fig. 8, c and d). Cover measurements with glasses were 9△ esotropia for distance; 23△ esotropia for near. Without glasses the measurements were 34△ esotropia for distance; 36△ esotropia for near. Second-grade fusion was found at the subjective angle of 9△ base out. The eyes were crossed for distance and looked even worse for near. Since the deviation appeared to be undercorrected, especially for near, it was felt in the clinic that a recession of one medial rectus was indicated to get the eyes straight. The attending surgeon in this case felt that even if an overcorrection was obtained, wearing of glasses could be discontinued and the result would be good. Too little attention was paid to the finding that this patient had only 5 degrees of esotropia with glasses, both with objective and subjective measurements.

On January 21, 1948, a 5-mm. recession of the left medial rectus muscle was made. Immediately after the operation the eyes were straight with glasses, but when cover measurements were taken, exophoria to exotropia of 13△ was found with glasses. The subjective angle was not investigated (since at that time we did not know its importance in determining what strength of glasses should be worn) and the correction was reduced by +.50 sphere empirically. The patient continued to wear a +2.50 sphere in each lens until December 1950, two years after the operation. This must have been too strong, for at that time a definite exotropia was observed. The cover measurements with glasses were 13△ exotropia for distance; 9△ exophoria for near. Without glasses they were 2△ esophoria for distance; 13△ esotropia for near. Wearing the glasses was discontinued.

After going without glasses for one month the patient was examined again and the eyes were found to be straight for distance and to have 15 degrees of esotropia for near (fig. 9, a and b). This apparently was a good result for distance, but the problem was what to do about the esotropia for near. Second-grade fusion was found at the subjective and objective angle of 2△ base in. This was of help, since it indicated that an exotropia was present for distance without glasses even though an esophoria was found with prism cover measurements. Glasses were not given for the esotropia at near because we wanted to see whether we could prevent the exotropia at distance from getting worse. Unfortunately, the exotropia progressed without glasses, and when the patient was examined in December 1955, seven years after the second operation, he had 20 degrees of exotropia for distance and 10 degrees for near. (fig. 9, c and d). The cover measurements were 20△ exotropia, 4△ left hypertropia for distance; 18△ exotropia, 3△ left hypertropia for near. Second-grade fusion was now found at the objective and subjective angle of 24△ base in, 4△ left hypertropia. He has had two operative procedures for the exotropia, and it is not yet corrected.

From a study of this case, the following inferences can be drawn: (1) Surgical treatment of strabismus may produce overcorrection if the patient has a small subjective angle of squint. (2) When exotropia and esotropia are present in the same patient, one need not worry about correcting the esotropia. The tendency is for progression of the exotropia, so that in time the esotropia may be replaced by an exotropia. (3) Allowing the patient to continue to wear plus lenses without frequent determination of changes in the subjective angle may contribute to the production of an irreversible exotropia.

Case 6. A 3-year-old girl came to the Infirmary in November 1946, with a history of having had crossed eyes since she was 8 months of age. With atropine cycloplegia, results of refraction were O.D., +3.00 sph., visual acuity 20/30; O.S., +2.50 sph., visual

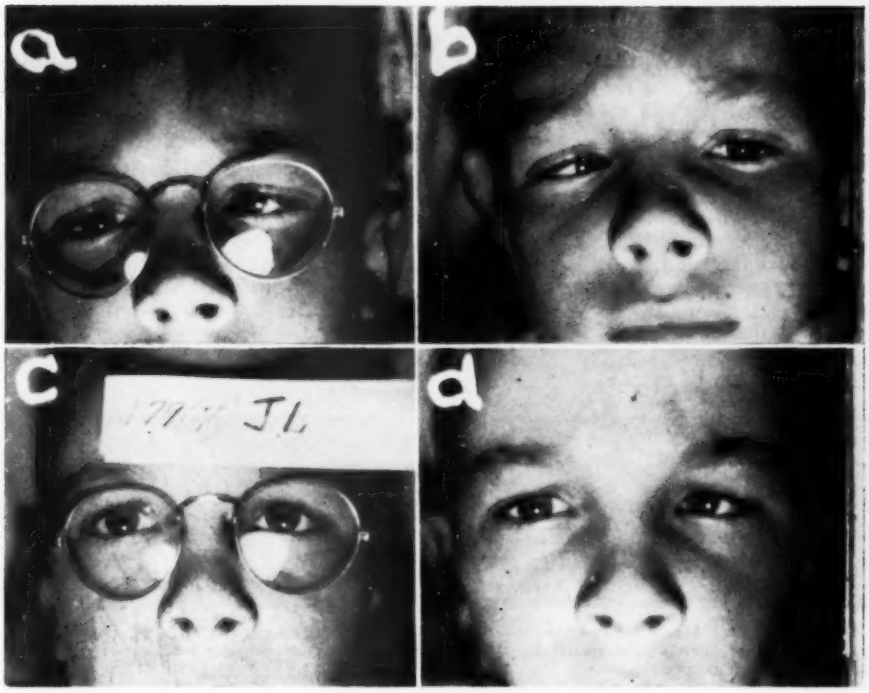

FIG. 8 – Case 5. (a and b) August 1947, 25 degrees of right esotropia with glasses; 30 degrees of right esotropia without glasses. (c and d) Two months after first operation, about 5 degrees of left esotropia with glasses; 20 degrees of left esotropia without glasses.

FIG. 9 – Case 5. December 1950, two years after second operation: (a) eyes can be straight without glasses for distance; (b) 15 degrees of right esotropia without glasses for near. December 1955, (c) 20 degrees of left exotropia without glasses for distance; (d) 10 degrees of left exotropia for near.

acuity 20/30. Examination revealed about 25 degrees of esotropia with glasses and 30 without (fig. 10, a). Cover measurements with glasses were 25△ esotropia for distance; 25△ esotropia for near. Without glasses the measurements were 35△ esotropia, 4△ right hypertropia for distance; 38△ esotropia for near.

On May 6, 1948, a 6-mm. recession of the left medial rectus muscle and a 9-mm. resection of the left lateral rectus muscle were done. Postoperative examination, 4 months later, revealed that quite a large amount of esotropia remained: 15 degrees with glasses and 20 degrees without (fig. 10, b and c).

FIG. 10 – Case 6. (a) May 1948, preoperatively, 30 degrees of right esotropia without glasses. (b and c) September 1948, four months postoperatively, 10 to 15 degrees of right esotropia with glasses; 20 degrees of right esotropia without glasses.

Another operation was suggested, but the mother refused to have it done. She stated that the child's eyes looked straight, especially when she was relaxed at home.

Examination in July 1950, two years after the operation, showed that there was about 10 degrees of esotropia with glasses and that 25 degrees could be present without glasses (fig. 11, a and b). Cover measurements with glasses were 18△ esotropia for distance; 27△ esotropia for near. Without glasses the measurements were 24△ esotropia for distance; 35△ esotropia for near. Second-grade fusion was present at the subjective angle of 10△ base out.

Examination in May 1956, eight years postoperatively, revealed that the eyes could be straight with and without glasses (fig. 11, c and d). Cover measurements with glasses were now 6△ esotropia for distance; 12△ esotropia for near. Without glasses, the measurements were 16△ esotropia for distance; 12△ esotropia for near. Second-grade fusion was now found at 6△ base in.

In this patient, a large esotropia remained postoperatively and a second operation was not performed because the parents observed the small basic angle that was present. This was fortunate because the eyes have subsequently straightened. Not only have they become straight but a subjective angle of 6△ base in was found at the last examination, indicating an exotropia with glasses. The strength of the lenses will have to be reduced. A large exotropia would probably have developed had we performed a second operation on this patient as we did in the previous case.

Case 7. A 5-year-old boy came to the Infirmary in July 1949, with a history that his right eye had turned in since he was 2.5 years of age. Results of refraction with atropine cycloplegia were O.D., +4.50 sph., visual acuity 20/30; O.S., +4.50 sph., visual acuity 20/20. Examination revealed about 20 to 25 degrees of esotropia with and without correction (fig. 12, a and b). Cover measurements with glasses were 44△ esotropia, 2△ right hypertropia for distance; 52△ esotropia, 3△ right hypertropia for near. Without glasses the measurements were 63△ esotropia, 4△ right hypertropia for distance; 70△ esotropia, 3△ right hypertropia for near. Second-grade fusion was present with the troposcope at the objective angle, which indicated that a large squint was present most of the time and that surgical correction was necessary. Normal retinal correspondence was present.

On November 9, 1949, a 5-mm. recession of the right medial rectus muscle and an 8-mm. resection of the right lateral rectus muscle were done. At postoperative examination, one year later, the eyes were straight with glasses and had 5 to 10 degrees of esotropia without glasses (fig. 12, c and d). Cover measurements at this time with glasses were 5△ exophoria, 1△ left hypertropia for distance; 2△ esophoria, 2△ left hypertropia for near. Without glasses the measurements were 2△ exophoria, 1△ left hypertropia for distance; 14△ esophoria, 2△ left hypertropia for near. Second-grade fusion was now present at zero; normal retinal correspondence was present.

The child's condition was followed in the clinic at six-month intervals. When seen in April 1955, six years postoperatively, 10 degrees of exotropia was present with glasses

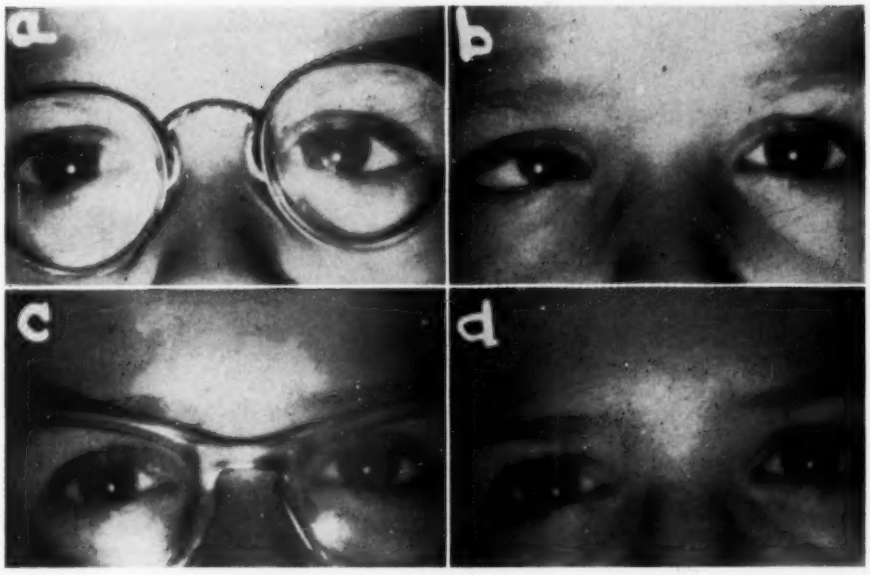

FIG. 11 – Case 6. (a and b) July 1950, two years postoperatively, about 5 to 10 degrees of left esotropia with glasses; 25 degrees of right esotropia without glasses. (c and d) May 1956, eight years postoperatively, eyes straight with and without glasses.

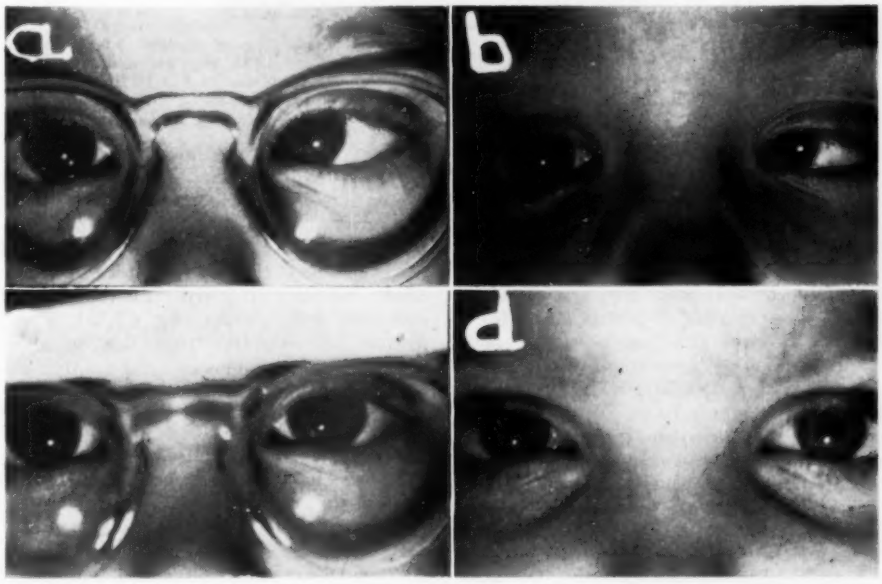

FIG. 12 – Case 7. (a and b) July 1949, about 20 to 25 degrees of left esotropia with and without glasses, preoperatively. (c and d) November 1950, one year postoperatively, eyes were straight for distance with glasses; 10 degrees of right esotropia without glasses.

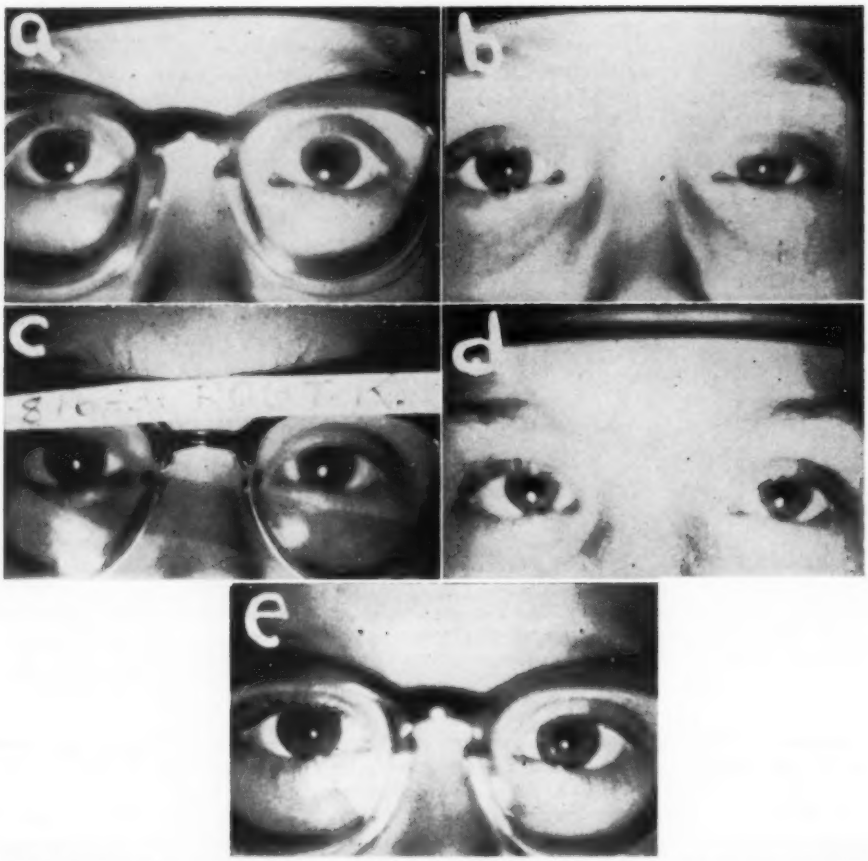

FIG. 13 – Case 7. (a and b) May 1955, six years postoperatively, 10 degrees of right exotropia with glasses; 5 degrees of right exotropia without glasses. (c and d) June 1955, after reduced correction had been worn for one month, about 5 degrees of right esotropia with glasses; about 10 to 15 degrees of right esotropia without glasses. (e) May 1956, about 5 degrees of right exotropia with glasses.

and about 5 degrees without (fig. 13, a and b). The NPC was 40 mm. Cover measurements with glasses were now 8△ exotropia, 1△ right hypertropia for distance; 12△ exotropia, 1△ right hypertropia for near. Without glasses the measurements were 5△ esophoria, 1△ right hypertropia for distance; 4△ esophoria, 1△ right hypertropia for near. Second-grade fusion was present at the subjective angle of 18△ base in. The correction was reduced from +4.50 to +3.50 sph. in each lens. One month later, examination revealed that now a definite esotropia was present, 5 degrees with glasses and 10 to 15 degrees without (fig. 13, c and d). The cover measurements with glasses had changed to 8△ esotropia for distance; 8△ esotropia for near. Without glasses the measurements were 23△ esotropia for distance, 23△ esotropia for near.

It was necessary at this time to decide whether the strength of the lenses should be increased or whether the weaker lenses should be given a further trial. The decision to keep the weaker lenses was based on the finding of second-grade fusion at zero. This decision, based on the subjective angle, was proved correct when the patient returned to the clinic one year later with about 5 degrees of exotropia with glasses (fig. 13, e). The subjective and objective angle had changed to 4△ base in with glasses, and another reduction in the lenses was indicated.

Case 8. A 5-year-old boy came to the Infirmary in September 1943, with a history of his eyes having been crossed since birth. Results of refraction with atropine cycloplegia were O.D., +1.50 sph., +2.75 cyl., axis 100, visual acuity 20/30; O.S., +2.00 sph., +3.25 cyl., axis 80, visual acuity 20/200. With occlusion of the right eye, vision in the left improved to 20/40.

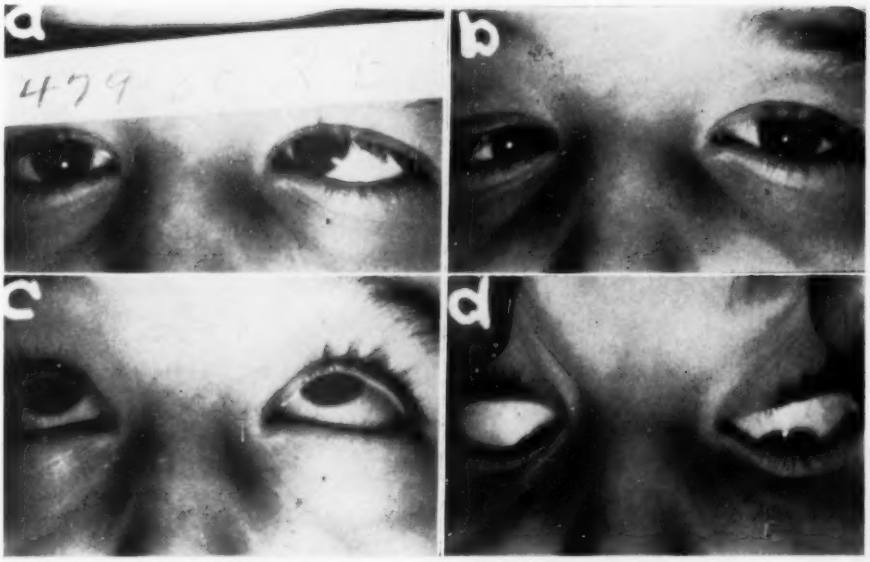

FIG. 14 – Case 8, January 1951. (a) 30 degrees of left esotropia; (b) 30 degrees of right esotropia; (c) about 5 degrees of esotropia on looking up; (d) 35 degrees of right esotropia on looking down.

Examination in January 1951, revealed 20 degrees of esotropia with glasses and about 30 without glasses (fig. 14, a, b, c, and d). About 5 degrees of esotropia was present on looking up and 35 degrees on looking down. Cover measurements with glasses were 45△ esotropia, 3△ left hypertropia for distance; 54△ esotropia, 3△ left hypertropia for near. Without glasses the measurements were 50△ esotropia, 3△ left hypertropia for distance; 63△ esotropia, 3△ left hypertropia for near. Second-grade fusion was present at the subjective angle of 40△ base out on the troposcope. On January 15, 1951, a 5-mm. recession of the left medial rectus and an 8-mm. resection of the left lateral rectus were done. Postoperative examination, two months later, revealed about 15 to 20 degrees of exotropia for distance with glasses and 10 degrees of right esotropia without glasses (fig. 15, a, b, c, and d). There was 20 degrees of exotropia on looking up and 20 degrees of esotropia on looking down. Cover measurements with glasses were 15△ exotropia for distance; 10△ exotropia for near. Without glasses the measurements were 5△ esotropia for distance; 10△ esotropia, 6△ left hypertropia for near.

This result was very interesting. With such a large exotropia on looking up and an esotropia on looking down, what basic deviation did the patient actually have? This was determined to be an exotropia with the finding of second-grade fusion at 4△ base in, and the lenses were weakened to the cylindrical correction only. The change in the glasses made a marked difference in the deviations. When last examined in June 1954, the eyes were straight without glasses (fig. 15, e, f, and g). (At this time the glasses were worn for reading only.) Only about 5 degrees of exotropia was now present on looking up and the eyes had become straight on looking down. The cover measurements with glasses were orthophoria for distance; 3△ esotropia for near. Without glasses the measurements were 4△ esophoria for distance; 3△ esophoria for near. The objective and subjective angle was at zero. The patient had stereopsis at zero, with amplitude to 20△ base out.

It is interesting that with the development of good fusion the marked deviations that were present on upward and on downward gaze became much decreased. In this case, as in the previous one, the large subjective angle of deviation indicated that surgical intervention was necessary, and the postoperation results were good in both cases.

Case 9. A 6-year-old girl came to the Infirmary in November 1948, with a history of the right eye turning out since birth. Results of refraction with atropine cycloplegia were O.D., +1.25 sph., visual acuity 20/30; O.S., +1.00 sph., visual acuity 20/20. There was right exotropia of 30 degrees for distance, although the eyes could be straight for near (fig. 16, a and b). Cover measurements were 37△ exotropia, 9△ left hypertropia for distance; 25△ exophoria, 4△ left hypertropia for

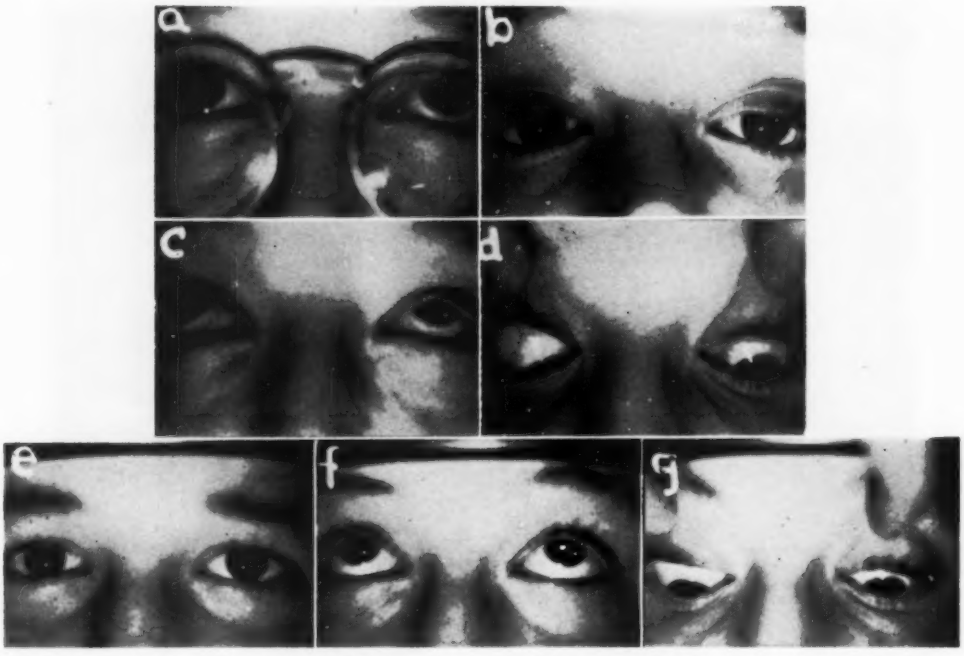

FIG. 15 – Case 8. March 1951, two months postoperatively, (a) 20 degrees of exotropia for distance with glasses; (b) 10 degrees of right esotropia without glasses; (c) 20 degrees of exotropia on looking up; (d) 20 degrees of right esotropia on looking down. June 1954, (e) eyes straight without glasses (glasses worn for reading only); (f) about 5 degrees of left exotropia on looking up; (g) eyes straight on looking down.

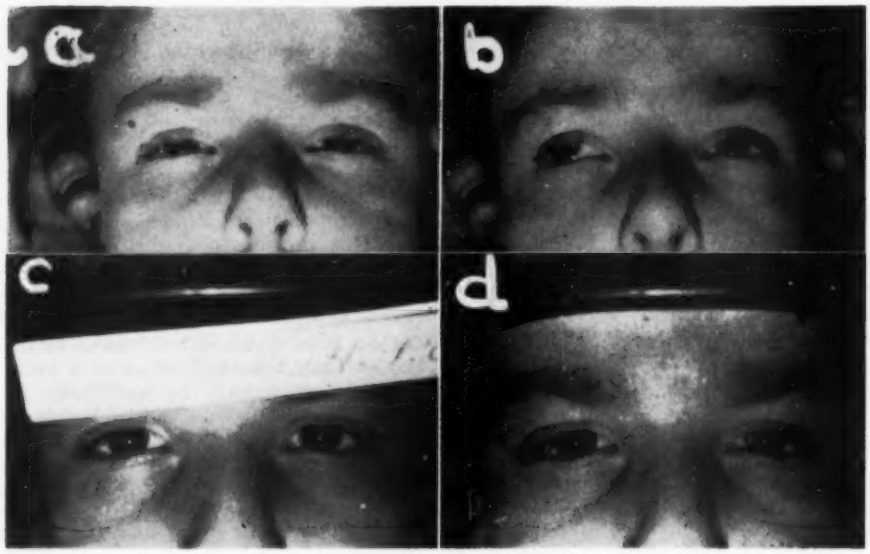

FIG. 16 – Case 9. November 1948, (a) straight eyes for near; (b) 30 degrees of right exotropia for distance. Three months postoperatively, (c) eyes straight for distance; (d) eyes straight for near.

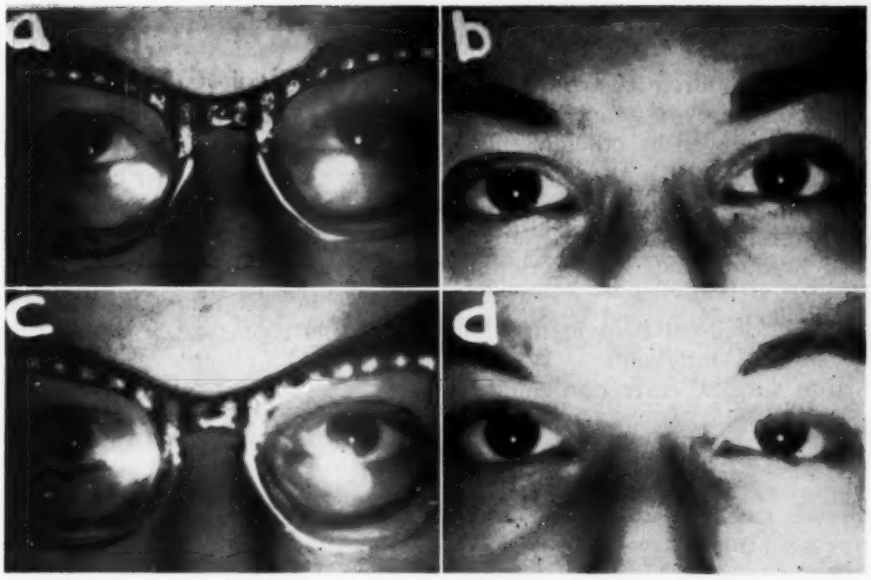

FIG. 17 – Case 9. (a and b) March 1955, seven years after first operation, 20 degrees of exotropia for distance with glasses; eyes straight for near without glasses. (c and d) Six months after second operation, 15 degrees of right exotropia for distance with glasses; eyes straight for near without glasses.

near. The subjective-angle findings were very interesting in this case. Although the eyes were crossed for distance, there was only second-grade fusion at the objective angle. For near, where the eyes were straight, third-grade fusion was found at zero. Note the decrease in the hypertropia as the child fused. There was a left hypertropia of 9△ for distance and 4△ for near and no hypertropia when fusion was not disturbed.

On November 15, 1948, an 8-mm. recession of the right lateral rectus muscle and a free tenotomy of the left lateral rectus muscle were done. Three months later the eyes were straight for both distance and near (fig. 16, c and d). Now third-grade fusion was found for both distance and near on the troposcope at zero, with good amplitude. The cover measurements were orthophoria for distance, 3△ exophoria for near. The hypertropia had completely disappeared, showing that it was a secondary vertical deviation (see discussion of case 2).

At re-examination in March 1955, seven years after the operation, it was found that the exotropia for distance had recurred (fig. 17, a and b). The refraction had changed from +1.00 sph. to −1.75 sph. for each eye. There was 20 degrees of exotropia for distance, but the eyes were straight for near with glasses. Cover measurements for distance were 20△ exotropia, 6△ left hypertropia for near; 14△ exophoria for near. Second-grade fusion was present at 28△ base in, 2△ left hypertropia. Note that with the recurrence of the squint for distance the hypertropia reappeared and, more important, stereopsis was lost.

On January 24, 1956, a 6-mm. recession of the right lateral rectus muscle was performed. Postoperative examination, six months later, revealed that about 15 degrees of right exotropia could be observed for distance, although the eyes appeared straight a good deal of the time. For near, the eyes were straight (fig. 17, c and d). The cover measurements with glasses were 6△ exotropia, 4△ left hypertropia for distance; orthophoria for near. According to the objective measurements this appeared to be a rather good result. Yet when the subjective angle for distance was measured, second-grade fusion was present at 18△ base in, 5△ left hypertropia; for near, third-grade fusion was present at zero with −3.00 sph. lenses. The subjective angle, being greater than the cover measurements for distance, possibly indicated a tendency for the exotropia to progress. This will be watched, and if the exotropia becomes greater, surgical correction will have to be attempted again.

SUMMARY

The subjective angle of deviation in cases of concomitant squint with normal retinal correspondence has been

shown to have had the following practical applications:

1. In some children it gave the basic deviation of the eyes, which could not be obtained as well by other methods.

2. It indicated what the position of the eyes would be over the long term even though the day-to-day deviations, as seen in the clinic, were very fluctuant.

3. It was of great help in deciding about surgical intervention. When it was large, surgical correction could be attempted with confidence. When it was small, unnecessary operations, with subsequent overcorrection, were avoided.

4. It was of great help in cases with a large accommodative component in assuring that the proper correction was worn. It indicated when the strength of the glasses had to be reduced or increased.

5. It helped to determine whether the hypertropia was a primary deviation due to a paretic vertical muscle or was secondary to the horizontal squint.

USE OF PRISMS IN THE TREATMENT OF PHORIAS

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SURGICAL treatment does not play an important role in the management of the various phorias as relatively few patients so afflicted are subjects for surgical intervention and only a small portion of those needing it will consent to surgical correction. Orthoptic measures, either of a formal type or in the nature of exercises at home, are of real value in the treatment of convergence insufficiency, but often they are of value in the other phorias only when used with supplementary measures.

Proper treatment must be preceded by diagnosis and classification. The most commonly used classification is based on whether the phoria is greater with distant fixation than it is with near fixation. Phorias that are greater with near fixation are assumed to be primarily anomalies of convergence, while those greater at distant fixation are anomalies of divergence. Experts disagree as to whether or not an actual divergence mechanism exists. However, recent electromyographic studies have given further support to those who believe in active divergence. Thus, abnormal exophoria is considered to be a convergence insufficiency if it is greater at near fixation, whereas it is considered to be a divergence excess if it is greater at distance. On the other hand, abnormal esophoria is considered to be a convergence excess if the measurement is greater at near fixation, while if it is greater at distance, divergence insufficiency is postulated. These criteria cannot be taken too literally, however, for a difference in phorias normally exists depending on whether they are measured at 20 feet or 15 inches. A commonly accepted normal limit for phoria at distance is 4 prism diopters of esophoria to 4 of exophoria, while at near fixation, the limit is 1 or 0 prism diopters of esophoria to 7 or 8 of exophoria.

The difference between the distant phoria and the near phoria is a variable amount, but few textbooks explain why it varies or on what functions it depends. From a purely mechanical-optical standpoint, this difference in phoria can be calculated. For example, a patient is orthophoric at 20 feet, at which distance it is assumed that the visual axes are parallel. If this pair of eyes should take up fixation at 0.33 meter, the eyes will have converged 3 meter angles. This convergence is converted to prism diopters by multiplying it by the pupillary distance in centimeters. Thus, for a pupillary distance of 6.7 cm., the near phoria would be 20 prism diopters. This is far greater than the normal upper limit and would be a most unusual finding. The factors that usually operate to reduce this figure are the accommodative convergence and, to a lesser degree, the proximal convergence. It is arbitrarily stated that the accommodative convergence serves to reduce this figure by about one-half, and the proximal convergence reduces it further by about one-fourth. Application of these factors then reduces the near phoria to about 4 or 5, which is within the normal limits. The activity of these compensating mechanisms is

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variable, thus accounting for the variability of the relation between phoria for distance and for nearness. Fusion, or fusional convergence, makes up the remainder of the adjustment necessary for maintaining single binocular vision. It is apparent, then, that accommodative convergence is a potent factor in maintaining a low exophoria at near fixation and that the decrease of accommodation with age accounts for the higher exophoria commonly associated with presbyopia.

Other factors are operating that complicate even more the understanding of the behavior of the eyes in convergence and divergence. Mechanical factors, such as resistance and inertia, as well as physiologic factors like fixation disparity, also play a part. Thus, the treatment of phorias cannot be generalized, as it is evident that abnormal phorias of equal amount may be caused by different factors.

Fusional convergence probably is the mechanism that is most flexible, and that is the one factor responsible for the fine adjustment necessary. Lessening of accommodation with presbyopia and the subsequent correction with plus lenses may lead to greater exophoria at near fixation. However, symptoms from this are not common because of the increased fusional convergence that can be brought into action. The limit to which all the convergence factors can be taxed can be measured by prism convergence studies; the opposite, or divergence, functions can be measured similarly. The breakdown into the different components, however, is almost impossible. Partly because of this, complete reliance cannot be placed on the findings of forced prism vergences, either the break point or the recovery point, in determining the proper treatment of phorias.

Use and ability fractions and comfort ratios have been proposed to help in the management of phorias. They are of aid in some cases, but they cannot be applied universally. Some authors have stated that one-third to one-half of the vergence power must be kept in reserve in order for a phoria to be asymptomatic. This is a good rule of thumb, but, like all rules, it has many exceptions.

The question remains as to how one can treat the phoria in cases in which measurements and rules do not always apply. The primary problem arising in a patient who has ocular discomfort is the determination of its cause. Careful refraction usually serves to ascertain whether or not a refractive error is the cause. A considerable number of patients have relatively small refractive errors, correction of which will bring comfort to the patient. In many other patients of the same age and with the same small error, such correction makes no difference whatever in their ocular comfort. The same individual variations are present in patients who have heterophorias. Every day we see patients who have exophorias of 15 prism diopters and who do book work with comfort, whereas other patients are miserable with small exophorias even though the vergences and refractive correction may be identical. Most ophthalmologists do not hesitate to prescribe a refractive correction when the need may be questionable, and they do this on the basis of trial. The patient cannot be assured of complete relief of symptoms when the ophthalmologist has no way to be sure that the symptoms are due to the refractive error. Often the patient is told that a certain error exists and that the correction may or may not relieve his symptoms. A trial period with glasses is the only way of being sure one way or the other.

A similar trial correction with prisms for the patient who has heterophoria has been used for years and appears to be gradually gaining in acceptance. Actually, this type of procedure is eas-

ier to carry out than is a similar trial with corrective power lenses. This procedure is almost as easy for those ophthalmologists who dispense glasses as it is for those who send patients to a dispensing optician. In either case, a few pairs of round meniscus plano prisms of various strength, for example, 1 through 5 prism diopters, are made available in clip-on mountings. Round rims make it possible for the prism to be oriented with its base in any direction by loosening the ring setscrew and turning the lens. The lenses should have the base clearly marked. If, for example, one wishes to test the effect of 1 prism diopter, base down, in front of the right eye for a small right hyperphoria, the suitable prism can be rotated properly in the right side of the frame, the left lens removed and the clip-on frame attached to the patient's glasses for a few days. Similarly, a patient who has 18 prism diopters of exophoria can try the effect of 4 prism diopters, base in, in front of each eye; if the exophoria is present only for near fixation, the clip-on prism can be used only for reading. If the desired results are forthcoming after the trial period, the prism can be incorporated in the patient's corrective lenses without uncertainty as to its effect. This trial method also is applicable in determining the minimal strength of prism that is effective after it has been proved that prisms are desirable.

The very mention of prisms to be worn before the eyes has, in the past, brought out dire warnings from some ophthalmologists as to the harmful and highly deleterious effect on muscular balance. The most frequent admonition was that once the patient wears prisms he cannot be without them and the prisms constantly must be increased in strength. These warnings are not entirely without foundation, but the implications need proper evaluation. If a patient has complaints referable to asthenopia and is found to have considerable hyperopia, there is no question but that he should be fitted with plus lenses to relieve the complaints. One could say with complete justification that once this patient starts wearing plus lenses he will not be able to go without them with comfort, and in all likelihood, as he grows older the plus lenses will have to be increased in strength. Nevertheless, one does not hesitate to give the patient lenses for either reading or constant use. Similarly, if prisms relieve complaints, there should be no compunction about prescribing them.

Patients who have hyperphoria usually are the easiest to prescribe for. Once the condition is corrected, it usually changes but little. This probably is because the vertical compensatory mechanism operates at a greatly reduced level compared to the horizontal. Vertical prism vergences are low, and vertical phorias usually are more stable than are horizontal phorias. Hyperphoria of 1 prism diopter or more should be considered for correction if symptoms exist, although hyperphorias of 2 prism diopters or more are perfectly compatible with comfortable vision. The vertical prism vergences often give a hint as to what degree of hyperphoria should be corrected in the trial prisms.

For example, in a patient who has a right hyperphoria of 3 prism diopters, with a base-down vergence of 5 prism diopters, and a base-up vergence of 1 prism diopter, it would appear that 2 prism diopters, base down, for the right eye would balance the vertical vergence. This is only a two-thirds correction, but allowance should be made for compensation developed over the years; full correction in this case would throw the vergence balance in the opposite direction, thus creating a left hyperphoria of a sort. With larger degrees of hyperphoria, it frequently works out fairly

well to correct only that part of the hyperphoria required to restore single binocular vision when looking at the test letters in a darkened room. Consideration of diplopia is not strictly part of a discussion of phorias, but some of the small vertical tropias with diplopia are handled in the same manner as are phorias. For example, if a patient presents 8 prism diopters of right hyperphoria with diplopia, the vergences cannot be measured. One tries to avoid putting on more prism than is actually necessary. In such a hypothetic case, it is possible that with 4 prism diopters, base down, before the right eye, single vision for objects in an examining room might be restored, but likely only by exertion of all the fusional mechanism at the patient's command. If the room is darkened and peripheral fusional stimulation removed, somewhat more prism will be required but likely not so much as if a red glass were used, which has been advocated in the past. It is common for a patient with a completely asymptomatic phoria to experience diplopia when looking at a light through the red glass, and if ophthalmologists use the abolition of diplopia with the red glass test, an unnecessarily strong prism often will be prescribed.

In correcting horizontal phorias, one gets into a much more controversial field. The question arises as to which of the exophorias at near fixation, the convergence-insufficiency type, should be treated by prisms, which should be treated by orthoptics, and which should be treated surgically. Generally speaking, the difficulty should be corrected with prisms in persons more than 40 to 45 years of age if a trial has shown that prisms offer relief of symptoms. Orthoptic training is to be preferred for younger persons. Surgical intervention is indicated for patients who have large degrees of phoria and who cannot be made comfortable with the usual prisms. If the contemplated prismatic correction does not overcorrect the exophoria for distance, the prisms can be ordered as part of the entire correction. If the exophoria is almost nil at distance, then the glasses should be ordered for reading only; or if the patient is presbyopic, prisms should be put in bifocal segments.

The next commonest group of patients for whom prisms are indicated are those who have esophoria at distance, the divergence—insufficiency group. Exercise often is of little avail in this group, and if prism trial indicates that the defect is too great for this type of correction, surgical treatment frequently is indicated. This type of phoria is associated more commonly with systemic illness than are the others, and one should not be too hasty in suggesting surgical treatment. Spontaneous recovery or improvement is the rule rather than the exception, and I prefer to observe such patients for nine to twelve months before suggesting surgical interference.

In this divergence-insufficiency group, the esophoria at near fixation often is absent and prismatic correction is necessary only for distant fixation. Two pairs of glasses frequently are necessary, although some patients prefer to use the clip-on arrangement for such activities as driving and movies, removing the prisms the remainder of the time.

An excess of divergence seldom calls for any prismatic correction. This condition usually is asymptomatic in its smaller degrees, while the complaint of patients who have larger amounts of exophoria at distant fixation is that one eye wanders out when fixing on distant objects. With exophoria of 20 prism diopters or more, the best results are obtained by surgical correction. Orthoptics offers some chance of improvement in patients who have lesser degrees of exophoria at distance.

Patients with an excess of convergence and esophoria at near fixation often can be made comfortable by full correction of the refractive error, but at times overcorrection by use of reading glasses or bifocals must be resorted to. Prisms can be used at times for patients who cannot be made comfortable by other optical means and in whom contraindications to their use are absent. However, a word of caution is in order because the prism must be reduced to maintain comfort as these patients grow older and become presbyopic. Again, it is a matter of a trial period to prove whether or not prisms will achieve comfort for the patient.

SUPPRESSION

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THE purpose of this paper is to emphasize the importance of diagnosis and treatment of suppression in children with strabismus. I wish to give an outline of things which can be done and which are practical in our treatment of suppression. This treatment is mainly an orthoptic problem; therefore, I have made an effort to include many orthoptic procedures and techniques. This has been done because the practical aspects are less well covered in the literature than are the theoretical aspects. It is hoped that this paper will act as a guide to which may be added any other methods with which the reader may be acquainted. I sincerely hope that new interest in the treatment of suppression will be aroused. Logically, the treatment of suppression is the backbone of all fusion training.

Suppression may be defined as the inhibition of incompatible patterns in the visual intellect. The entire retina is continually being bombarded by images of objects in our environment. Were it not for the inhibition of all images except those that arouse the visual intellect, a state of confusion would exist that would be incompatible with our existence.

When the visual axes are so malaligned that fusional amplitudes cannot make adequate adjustment, a heterotropia occurs and diplopia results. This is unacceptable to the individual, and he subconsciously makes use of suppression to rid himself of the problem. Before the advent of modern ophthalmology this was the desirable solution in obtaining single vision, but today the ophthalmologist has at his command the tools whereby he can usually improve the alignment, so the development of suppression is no longer the only solution. When alignment can be improved, abnormal suppression must be overcome before good fusion and fusional amplitudes are attained. Suppression is one of our most important clinical problems. It is the sensory barrier to be overcome after amblyopia and anomalous correspondence have been eliminated and before fusional amplitude training is started.

The sensory adaptations to strabismus develop progressively as loss of retinal rivalry, suppression, amblyopia, and/or anomalous correspondence occur. At whatever level of adaptation we find the patient, we must start treatment and attempt to reverse the sequence of events. Today, fortunately, fewer cases are neglected to the point that amblyopia and anomalous correspondence are deeply established. Nevertheless, nearly all of our patients show suppression of varying degree and intensity. Often its presence is not easily detected by amblyoscopic examination because of the artificial nature of the test.

Let us consider the development of suppression in strabismus, since treatment should be based mainly on its presence. As suggested before, when squint occurs, assuming that single binocular vision was present before its onset, diplopia and confusion result. In strabismus, the two maculas are directed at different objects, and these

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objects are visualized superimposed, causing *confusion. Diplopia* is caused by the visual image of the object of regard falling upon the macula of the fixing eye and on the extramacular area of the other eye, with visual projection of the same image in two separate spatial directions. We must remember that the maculas and the corresponding extramacular areas are not the only parts of the retina working together, but that *all parts of the retina of each eye are confronted with a similar problem,* although the patient is not aware that his entire visual field is affected by diplopia and confusion at the same time.

DIAGNOSIS

The detection of suppression and testing of fusion ability are of primary interest and concern in this presentation. There are several rules to be remembered. *Any patient who at any time or at any visual task has a heterotropia without discomfort has suppression. Likewise, any person who is made heterotropic artificially and has no complaints is suppressing.* Whenever possible, test under conditions which resemble everyday life. Test yourself carefully, and practice using the instruments and all methods which you use for treatment until they become very familiar. Simulate the problem in yourself when possible by inducing in yourself an artificial heterotropia. It is important that we remember what the average individual with straight eyes reports when he is artificially rendered heterotropic. This should be considered an important criterion in the evaluation of reports by a patient with a true squint whose suppression and other adaptations have been eliminated. I believe that the above points are more important when considering the patient's fusion ability; examination of patients to detect suppression should be carried out with these in mind.

Patients who have single binocular vision some of the time in daily life are more likely to gain from orthoptic treatment than those who never fuse casually. It is almost futile to treat patients in the latter group who are without complaints and whose squint is of long duration and of early onset unless the eyes are straightened by some means. If surgical correction is contraindicated in a case of constant tropia, treatment of suppression should be discouraged. Even though fusional ability on instruments may be present, the patient cannot use it in daily life, and practical orthoptic training cannot be carried out for periods sufficiently long to be helpful.

The presence of fusional amplitude relative to accommodation of sufficient amount to make the deviation latent is most important. If an amblyoscope is used, careful consideration should be given to determine that amount of amplitude which is relative, as amplitude generally increases under conditions of uncontrolled accommodation.

Points to be stressed in the determination of the presence or absence of suppression are as follows:

1. Secure fusion, that is, the ability to maintain straight eyes in spite of repeated attempts to dissociate them by various means, is a good indication that little or no suppression is present.

2. Suppression is present in constant and intermittent tropias which do not produce symptoms of diplopia or annoyance.

3. When the amplitude of fusion is checked, regardless of the method used (direct or indirect), diplopia should be noted immediately at the end point of the amplitude. If it is not, suppression is present. If there appears a slight shift before an obvious tropia is manifested at the end point of amplitude, central suppression should be suspected.

4. Suppression may be "intermittent," that is, it may be present under some conditions, in some fields of gaze, or at some distances while not present under other circumstances.

5. The intensity of suppression can vary from slight or fleeting to complete. Likewise, the area of suppression may be large or small.

6. When using instruments such as the amblyoscope, a good criterion for the absence of suppression is the ability to maintain fusion, first and second degree, on large and small targets for at least 20 seconds.

7. Strong suppression is indicated on instruments when one target seems to disappear or cross over the other as the objective angle is approached.

8. When suppression is present on using the red-green filter test, it is often fairly deep seated. If, on presenting a prism base down or base up before the suppressing eye, there is still no diplopia, the suppression may be very deep seated and the area of suppression large.

9. Very deep suppression may be found at times with the afterimage test, the patient failing to see one of the rods of light.

10. Further tests for suppression may be made by the use of bar reading, framing, or the diploscope, all of which involve physiological diplopia.

11. Tests of stereopsis, such as the Keystone DBI slide test or the Wirt stereotest, will give an indication of the depth of suppression, since patients with moderately strong suppression often do poorly on these tests.

TREATMENT

The treatment of amblyopia is beyond the scope of this paper. I should like to mention, however, that the final steps in obtaining equal binocular acuity and fixation should be persistently carried out whenever possible. Of course, this frequently is not attained in spite of our efforts. Nevertheless, that difference of the last line or two of acuity is a distinct barrier when fusion and especially fusional amplitudes are insecure and unstable.

The use of occlusion to overcome suppression in cases of amblyopia is a monocular procedure for the improvement of a condition established under a binocular situation. I do not feel that occlusion is a method for treating suppression, yet it may be considered as one method to use at times against suppression. *Principally, however, antisuppression exercises are done under binocular conditions, and there is no doubt that improvement can be achieved in many patients treated in this way.*

The presence of spontaneous or easy alternation when the patient is tested while reading the vision chart is a good sign that acuity is nearly equal in the two eyes (provided corrective lenses are worn if indicated). In any event, the teaching of alternation on command is a helpful orthoptic procedure as a first step in teaching diplopia and other exercises. Understandably, the patient should be able to maintain his attention and cooperate. The greatest aid in home treatment is for the patient with divergence to learn to control his eyes, that is, to manifest an exotropia on command and to straighten his eyes at will. Patients with accommodative esotropia should be taught to do similarly, that is, when the hyperopic correction is reduced or removed the patient should then be able to blur (relax accommodation) or to accommodate on command. In this way patients with either type of tropia will be able to practice diplopia with ease; they can be taught to appreciate diplopia through the entire range of deviation, from that at the moment bifoveal fixation is lost to the maximum angle of squint. Some patients with vertical deviations and some with incomitant strabismus will be found who can be taught to control their squint; these cases will benefit from instruction on control.

I. Diplopia

Control of deviation is frequently taught in conjunction with diplopia exercises to patients who show fusion some of the time in daily life. It should be understood that before giving diplopia exercises, we should make sure that the diplopia reported by the patient is normal rather than paradoxical. Patients who may be taught diplopia are in the following groups:

1. Most patients with exotropia-phoria, accommodative esotropia-phoria, or dissociated vertical divergence.

2. Some patients with mechanical esotropia or incomitant squint who show fusion some of the time in daily life. In these cases the exercises should be taught only if the ophthalmologist feels that surgical intervention is indicated to overcome an incomitance or to improve the basic alignment.

3. Patients not yet in their teens (and some older) who have fusion some of the time in daily life and whose alignment problem is not a barrier.

4. Patients under the age of 8 who show no fusion in daily life but are able to fuse under testing conditions, and who are to have a surgical correction.

Diplopia should be taught primarily with the dominant eye fixing, and ultimately with either eye fixing. Diplopia is usually taught in the following order:

1. On a light, then on an object, and finally, spontaneous and annoying diplopia whenever the patient is squinting.

2. At all distances, but especially at the distance and/or the field of gaze in which the deviation is most frequently manifested.

3. While the patient is changing accommodation, that is, maintaining diplopia while moving toward or away from fixated objects, or while voluntarily changing accommodation and amount of deviation.

The exact method to use in teaching diplopia (after Aleatha Tibbs) is outlined below. (These directions are given to the parent for guidance at home, after the exercise is demonstrated in the office.)

DIPLOPIA EXERCISES

(Technique)

The child must be able to hold attention on one object without moving his eyes for 30 seconds. His concept must be clear so that he can report whatever he sees.

Equipment

1. Light at 20 feet (cut a hole, one-half inch in diameter, in a box and place the box over a 30- or 40-watt bulb). Place this at the end of the room at the patient's eye level.

2. Hand light (a pencil-flashlight), or white-headed pin.

3. Red filter.

4. Opaque cover (small piece of pasteboard).

Steps

1. Seat the patient at an appropriate distance from the light. Alternate the cover from one eye to the other until you see that one of the eyes, as you uncover it, moves toward or away from the nose. Stop the cover over this normally nonfixing eye.

2. Place a red filter behind the cover. While the patient keeps constant fixation with the uncovered eye, cover and uncover the eye under the red filter until he is conscious of a red light blinking off and on. Continue this for ten to fifteen seconds, until he sees two lights, one red and one white, simultaneously; then gradually slow down to a stop, leaving the eye uncovered. If the red light disappears, repeat this procedure a few times to help the eye regain the red light.

3. When, with the red filter, the patient can see the two lights simultaneously and hold them for 30 seconds, the above process is repeated, using the red filter in place of the opaque cover; that is, cover-uncover the normally nonfixing eye until the light begins to blink alternately red and white. Continue for a few seconds, then gradually slow down until he sees two white lights simultaneously for 30 seconds with both eyes uncovered.

4. At this point, the patient must learn to tell which eye is straight and which is crossed. Both eyes are never crossed at the same time; one must be straight in order to see clearly. The straight eye sees a clear light; the deviated eye sees a dim light. He must see double regardless of which eye is straight; consequently, ask him to look at the light on the left side and, without moving his eyes, observe that it is clear and the light on the right side is dim. Then ask him to look at the light on the right side and, without moving his eyes, observe that it is clear and the left one is dim.

5. Double vision must be appreciated when the patient's eyes are out of line at all distances from 13 inches to 20 feet. At first, have him sit at an appropriate distance from the light, and when he can do step 4, have

him back away two or three feet at a time, maintaining double vision. If he loses one light, he must report it. Then repeat step 3 to help him regain double vision. When he sees two lights at 20 feet, have him walk slowly forward to a few inches in front of the light, making sure that he sees two lights all the way. The above procedure is to be practiced whether the patient is looking at a light, picture, word, or letter.

When diplopia is achieved by the above exercises, it is then wise to have the patient move his usually deviating eye from the position of fusion to that of maximum squint and return to fusion. The appreciation of diplopia through the *entire* range of deviation is wise. It cannot be overemphasized that this diplopia should be appreciated on objects in daily life as well as on special lights, pictures, words or letters. Patients showing noncomitance should practice fusion and recognition of diplopia as their squint becomes manifest in the field of greater deviation.

II. Physiological Diplopia

Achievement of physiological diplopia is another form of exercise and is helpful for the following reasons:

1. It gives the patient a way to check on himself.

2. It helps to stabilize fusion.

3. If properly practiced, it will do much to eliminate suppression.

4. Though it is normally not noted, most people with single binocular vision can learn it easily, yet those without single binocular vision usually cannot appreciate it.

This exercise can be given to patients who are able to fuse or who can be made orthophoric by lenses or by other means. We give physiological diplopia exercises whenever practical to those who are practicing diplopia.

The general principles are that while single binocular vision operates, the fixated object and objects within Panum's area are seen in single vision. Objects outside of Panum's area are seen in crossed or uncrossed diplopia, depending upon whether they are closer or farther away than the fixated object.

Before giving a patient this exercise for home work, as in diplopia, we try to give the parent a clear concept of what is expected. This is done by demonstrating the exercise on the parents themselves.

PHYSIOLOGICAL DIPLOPIA EXERCISE
(Technique)

The child must be able to hold his attention on one object without moving his eyes for 30 seconds. He must have fusion in daily life at some determined distance.

Equipment

Same as for diplopia exercises.

Steps

1. Seat the patient at an appropriate distance from the light. Do cover-uncover tests as an objective check for bifoveal fixation.

2. Place a red filter first before one eye and then before the other to see whether the child reports mixed light (fusion). The filter may be left before one eye if necessary, but it is best to remove it if it is not necessary. With the filter before the nondominant eye, the next step – seeing two lights either in front of or beyond the fixated light – may be more easily attained.

3. Introduce a small pencil-flashlight (or white-headed pin) either beyond or in front of the first light fixated. This second object should be seen double. If it is not, several things may be done to help in this achievement:

a. Move the pencil-light up and down or forward and backward.

b. Place a red filter before one eye to demonstrate physiological diplopia.

4. Physiological diplopia should be recognized under conditions of both distance and near fixation, that is, in crossed or uncrossed diplopia. When this can be achieved and maintained without suppression for 20 to 30 seconds, proceed with the next step.

5. While the patient keeps his eyes fixated on the stationary light, bring the pencil-flashlight closer to the stationary light, having him maintain and report diplopia until the fixed light and the double lights are quite close together.

6. The above procedures should be practiced next using such things as pencils, pins, and small objects, under conditions which approach normal everyday seeing.

7. Games and devices utilizing physiological diplopia may be employed in the final stages of the exercise. Examples of these are the mirror-star game, the extra-finger game, the target game, objects on glass through which the patient is looking, framing, bar reading, separator reading, diploscope, Remy separator, Javal grid reading, and hole in hand. (It is important that, whenever possible, we should simulate the squinting condition on ourselves in framing and bar reading. It is in this way that we can more fully appreciate the patient's problem.)

a. When the framing game is used (that of seeing a pencil in crossed diplopia on either side of the object of regard), it must be remembered that this can be done incorrectly by a patient with esotropia. The patient can see the pencils on either side of an object when suppression of homonymous diplopia takes place. If there is doubt, have the patient close one eye and then the other to observe whether the pencil touches each side of the object fixated. The pencil should be directly in line with the object fixated and the patient's nose. If framing is done incorrectly, the pencil will be closer to his nose than expected and off to one side. We all frame at approximately the same distance, and if we do the framing at the same time and at the same distance as the patient, this is another check on the accuracy of his framing.

b. Bar reading and/or separator reading should be carried out after framing is mastered. They provide the added advantage of a sustained antisuppression exercise in "seeing through" the bar or separator. We must guard against alternation, as some patients can read up to the bar, switch to the other eye, and read on without pausing. In this case, the eyes will jump as they travel along the line of reading to the bar. The patient must be taught to recognize his error.

III. Red-Green Filter Exercises

Remember that suppression is less likely to occur when colored filters are used. Therefore, as a first step toward fusion and elimination of suppression, this exercise is valuable. Nevertheless, discontinue use of the filters as soon as possible.

Place a red filter before one eye and a green filter before the other eye, or use only a red filter before one eye. Have the patient walk across the room toward a light (or some other white target) until the two images join and appear to have a mixed color. When this is achieved without suppression, the patient should slowly recede from the light, maintaining fusion without suppression. The goal in this exercise is to maintain a mixed light at all distances.

As a diplopia exercise, have the patient fixate one light with the dominant eye while seeing the other light as he changes his distance from the target and do so constantly without suppression. He may alternate on command and practice fixating with the other eye if alternate suppression is present.

Patients with deep suppression frequently will find this exercise difficult and be unable to fuse the images. The images may, in fact, "skip" over each other rather than fuse. Do not allow uncontrolled alternation.

The red-green filter exercise is of value as a control exercise. Many patients can learn to move their eyes to the straight position or to a divergent position, at will. This will aid us in applying exercises for diplopia and physiological diplopia without filters as a next step.

Physiological diplopia exercises may be done in conjunction with the red-green filter fusion and recession exercises.

With any kind of red-green filter exercise, the goal should be for the patient to be constantly aware of the two colors. This should be true whether he is fusing or not. When fusion is achieved, the patient must try to maintain it when either large or small targets are used.

IV. Filter Exercises

When suppression is present in the nondominant eye, although orthophoria is present and there is peripheral fusion, the use of red filter exercises is helpful. The filter should be used be-

fore the dominant eye, and care should be taken that the filter doesn't cause the eyes to dissociate, since they should remain orthophoric or nearly so. It should be remembered that a red filter is useful not only on red color but also on orange and yellow; in fact, it makes all colors appear different from normal. Therefore, the filter is helpful in any task or game involving colors. The use of a red filter with colors should involve "form sense," and the more detailed the task, the better, when central suppression is present.

There are several activities which may be carried on with the red filter, all of which may be presented to the patient as home work. Among these are the following:

1. Red writing and reading (Dobson reader). A simple way to vary the content is to type a story, using a red and black ribbon on the typewriter.

2. Tracing red pictures; red construction work.

3. Sewing, weaving, bead craft, picture puzzles, bead games, bingo, etc.

4. Putting red dot in small red circles (and needle in seed beads) for localization and fixation as well as for antisuppression.

5. Constant wearing, viewing technicolor movies.

V. Occlusion

1. Part-time occlusion consists of patching one eye or covering the lens of one eye some of the time; for example, for half a day.

2. Partial occlusion may take the form of strip occlusion, of covering part of the lens, of a coating of lacquer on the spectacle lens, or the use of Hamblins' Pugh graded occlusion lenses. The purpose of these devices is to provide a greater or lesser opportunity for some binocularity and to allow fusion while making the suppressing eye do a greater portion of the seeing. The chief benefit is perhaps derived when the seeing of form with the better eye is somewhat less acute than that with the suppressing and/or amblyopic eye. The less distortion produced by the partial occlusion method, the better.

VI. Cheiroscope, Tracograph, Pigeon-Cantonnet Stereoscope, Drawing Stereoscope, and Hand-Mirror Superimposition (Confusion) Game

These instruments are of considerable value in treatment of suppression. The eyes are dissociated, so that they are seeing two different objects. Since suppression is mainly a binocular condition and takes place while the master eye fixes (except in alternate suppressors), probably the most benefit would be derived while the master eye fixes. In opposition to this thought is the theory that the suppressing eye needs training in fixation, so it should be allowed to fix. There should be no objection to doing the exercises either way. They can be used in the following ways:

1. Tracing simple bold outline pictures and working toward detailed form tracing.

2. Crossing technique: fixating a small object, then endeavoring to insert it into a larger form, such as a circle, cage, or box. It is useful to have the patient fixate a dot while moving a circle back and forth several times before attempting to catch the dot in the circle.

3. Viewing a detailed picture while trying to insert a small object, such as a dot or symbol, on some part of it.

4. Placing a circle drawn on a small strip of paper either into or outside of a larger circle, or attempting to fuse it with a circle of the same size, on a sheet of paper with circles of various sizes on it. Many other types of targets can be used if one is imaginative and cares to go to the trouble to make them.

5. Playing the hand-mirror superimposition game is interesting to many children. They hold a hand mirror tilted before one eye and attempt to place an object seen by the other eye, upon an image in the mirror. The centers of both eyes must function together if this is to be achieved.

VII. Television Polaroid Trainer

For the last year and a half we have used the television polaroid trainer (American Optical Company) with more than fifty patients, but no study of the results has been made. I have the impression, however, that the trainer is particularly useful for patients with poor fusion and fair to good alignment. With these patients, we are striving to maintain good alignment in the hope that sufficient binocularity will be established to maintain this alignment. For patients with fair to good fusion with some suppression, I feel that active antisuppression procedures are better whenever practical. Nevertheless, the use of the television polaroid trainer as an adjunct may be helpful in the treatment of suppression.

VIII. Use of Stereoscope

Antisuppression exercises with stereoscopes at home are useful in the final stages when vergences are being practiced. Suppression is least likely to be present when there is diplopia at the break point of fusion, and when parts of the fused picture can be pointed to accurately on each side and check marks can be constantly noted. Finally, there should be stability of fusion, appreciation of stereopsis, notation of blur point, and good amplitudes of clear fusion.

IX. Office Training on the Haploscope

This is perhaps the most widely used antisuppression training. We are all familiar with crossing technique, shaking, fusion and break, catch game, etc.

SUMMARY

The purpose of treating suppression is to eliminate the final barrier to good and stable fusion and control of deviation. To leave suppression untreated is to court a breaking down of fusion and a tendency to return to the former deviation.

Before suppression can be eliminated the eyes must (1) be straight under some or all circumstances, and (2) have equal or nearly equal visual acuity.

It is a waste of time to treat suppression when the eyes have no opportunity to work together in daily life, since suppression is a binocular phenomenon, evincing itself only when both eyes are open. If suppression is overcome and the eyes are not straight at least intermittently, there results either an annoying diplopia or a re-established suppression.

I have endeavored to give the principles of diagnosis which are used in our office, and to list the methods which we use for treatment of suppression, with the hope that the reader will find herein inspiration for further effort, and that the subsequent discussion will help me in turn.

THE TELEVISION TRAINER IN FUSION TRAINING

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HOWEVER well-trained and competent the orthoptic technician may be, she will fail in her effort to establish binocular single vision in her patient unless she is a skillful teacher, for in the final analysis it is her teaching ability which produces the end result. There is, of course, a percentage of unavoidable failures because of poor cooperation on the part of the adult or child patient or of the parent in the case of a small child, and this lack of cooperation presents in many cases an insurmountable obstacle to the teaching orthoptist.

Since the end of December 1955, I have prescribed eighty-seven AO Polaroid Television Trainers, and I consider them to have been a successful adjunct to fusion training in 85 per cent of the cases in which they have been used. Because a considerable portion of my work is with children of Navy personnel, who are more or less transient, it is not possible accurately to assess the end results. However, we have been able to follow the cases of a sufficiently large number of these patients, as well as of patients who are permanent residents, so that we can state that the television trainer has proved its worth in our particular practice.

I shall not go into the mechanics of the television trainer, but rather shall concern myself with the proper use of the device.

Presented at the Annual Joint Meeting of the American Orthoptic Council and the American Association of Orthoptic Technicians, Oct. 14, 1956, Chicago.

I have in our clinic a dozen copies of the Scobee and Katzin book "Rehabilitation of a Child's Eyes," and parents of each patient are given a copy of the book to read. The parent, or both parents, will have observed the initial survey and become aware of his child's specific visual disability. He is instructed to pay particular attention to that portion of the book dealing with his child's problem. He is then told what we hope to accomplish by visual training. A sense of security must be developed in the child, so that he will give correct answers without fear of being reprimanded. All criticism on the part of the parent and the technician must be constructive. The child will soon work to merit praise, and progress will then be more rapid. Home training should be given as soon as possible so that the parent and the child will have a daily interest in the training and both can feel that progress is being made.

I believe that it is essential to transfer the binocular skill learned on the major amblyoscope and other instruments to casual everyday seeing as soon as it is possible to do so. If, however, home training is attempted too soon, both the parent and the child will become discouraged and further progress will be most difficult. There will be friction at home, and frequently behavior problems will develop. The wise orthoptic technician will first make certain that the child understands the procedure which she wishes carried out at home; then she will dem-

onstrate it to the parent. Finally, she will have the parent and child do the procedure together in the clinic until she is certain that they are capable of following her instructions at home. My patients are then given simple, typewritten instructions to which they may refer should they forget the oral instructions which they have received. I like to demonstrate all procedures on the parent, so that he or she may understand the effort and the amount of concentration required of the child in his home training.

The child is not ready for the television trainer until he can fuse and, above all, until he has confidence in himself and in his ability to fuse. His attitude must be positive rather than negative, since this is a home-training device which requires accurate subjective observations and second-grade fusion for successful application.

The patient is tested in the clinic on the trainer from three to five times before the parent is advised to purchase one for home use. The best results are obtained when the patient can fuse with effort at a distance of five to ten feet. It is important to determine the temperament of the parent and of the child, since an impatient, precocious child will obviously make less of an effort than will a patient, well-disciplined child who can be made to understand the value of the home training and in whom can be instilled the desire to cooperate in striving to obtain comfortable binocular single vision. The orthoptic technician sees the patient more frequently than does the doctor. She should be able to assess the psychological reaction of the child and of the parent to home training and to know when cooperation on the part of child and parent is possible. Some children are ready for the trainer sooner than others with an identical angle of squint because they are more mature emotionally or more intelligent or more cooperative. In working with children these are the factors which should be considered rather than the chronological age of the patient. Of course, the degree of attraction which television holds for the child is of major importance, since a child who enjoys television will progress much more rapidly than will the child who does not care for this form of entertainment. To know when to prescribe the trainer in a given case is perhaps the most important factor in the successful use of it and in the result obtained.

When I am ready to prescribe the trainer, I bring the parent in, seat him, or her, at the correct distance and demonstrate what the child sees when vision in the left eye is suppressed, what he sees when vision in the right eye is suppressed, what he sees when he alternates suppression, *how slight a head tilt is required to view the entire picture with one eye*, and finally, what the child should see when he is fusing properly. I then show the parent exactly how to apply the trainer and give him written instructions for reference at home.

I have found it advisable to begin with home-training periods of ten to fifteen minutes two or three times a day, with a gradual increase in time until the trainer can be used without too much discomfort for an hour at a time. When this goal is reached, most children will quite happily view most of their television programs with the trainer in position.

Before prescribing the trainer, I advise the parent of its cost. At the same time, I point out that when it is successfully used, the child is receiving daily therapy and clinical visits soon can be decreased, so it does save money in the long run. I also acquaint the child with the cost of the device, and the decision to use it is usually

made by him. He then takes a much more active interest in it than he would if he were simply told to sit down and use it.

I have frequently treated children who insisted that they could not use the trainer, although it was quite evident that they were fusing with it with some effort. While chatting with such a child, I asked him to spell the letters in an advertisement (this, of course, is not possible in preschool ages). When the child does so, he realizes that he has been trapped, and in the majority of cases there is no further difficulty. As with all methods of medical therapy, the approach to the patient must be a positive one – that is, what *he* is going to get out of it, since, after all, that is his primary concern. Once out of the clinic it is unlikely that he will continue his exercises just to please the technician, as he often does in the clinic.

All children look forward to the mid-teens, which they consider quite adult. I find that if I explain to them that the correction of the eye defect must be carried out while they are small so that when they are older and "date," or participate in sports, or apply for responsible positions, their eyes will not present a cosmetic handicap, they will make a much better effort than they might otherwise.

The trainer presents a pleasant means of home training whereby the patient can begin to know when his eyes *are* straight and when they are *not*. He will begin to feel the difference, and as fusion strengthens, the amplitude of fusion will increase to that point at which it is easier for him to fuse than it is for him to suppress, then deviate.

The orthoptist *cannot* expect to attain any degree of success with the trainer unless she has a television set and trainer in the clinic and can demonstrate it several times to the patient. I have my television set on rollers, and I begin working in the clinic at the distance at which fusion is most comfortably and easily obtained, gradually working in or out to the five or six feet advised by the makers of the trainer. I frequently use auxiliary prisms or lenses in order to encourage fusion with the trainer, but I do not prescribe it for home use until the patient can use it successfully while wearing his regular glasses.

I do not prescribe the trainer when the patient can easily sustain fusion at intermediate and far distances. Obviously such a patient does not need it, and ordinary viewing and games requiring binocular skill and depth perception are of more benefit. For these patients indoor quoits, or horseshoes, are very good, since the standards can be moved from near to far. Games such as tiddlywinks, in which the counters are flipped into a small glass, and any of the building or construction games are excellent. A Viewmaster stereoscope is very useful when fusion can be accomplished with it, and the toy shops are full of games which can be suggested. Once binocular single vision is possible at the near point, such games as pick-up-sticks are good. Bead stringing and sewing for girls and model building for boys should not be overlooked. These remarks are a bit afield from the subject of this paper; however, they should be borne in mind. As fusion develops and increases in amplitude with the use of the television trainer and training at the clinic, these games and others like them can be useful in reducing the duration and cost of treatment.

I have talked with many orthoptic technicians who stated that they are opposed to use of the trainer, only to discover that they have never used one

in their clinic. Orthoptics is a field which is constantly developing, and we should investigate every proposed new technique. After we have tried a method of therapy, we can reject it or not, as it proves or disproves its worth, *but it should be tried.*

When I prescribe the television trainer, I have the patient use it first with the label on the filter showing at the bottom of the picture. Then I advise him to turn the filter end for end every two weeks. This alternates the half of the picture which is suppressed; for example, if the right eye has been deviating in, and the right side has been darkened, the left side of the screen will now darken. I find that this procedure discourages suppression. Also, I like to cut the width of the central depolarization strip from three inches to one-half inch, since this area can be seen clearly with either eye separately. We have had some trouble with the suction cups on the filter, but we find that a mixture of glycerine and water makes them adhere better to the set and causes no damage to the finish of the set.

In checking with the Bureau of Internal Revenue I find that if a trainer is deemed necessary by the doctor and if the patient has no television set, or if his set is in need of such extensive repairs that a new one is necessary for successful use of the trainer, the cost of the set and the trainer is deductible as medical expense in the income tax report. If the patient has a television set in good working order, or one which can be put in good working order, only the cost of the trainer is deductible. The deduction must be listed in the itemized report together with the doctor's name and address.

Rather than present a statistical report, I have chosen to present several isolated cases – all problem cases – in which, I feel, benefit from the use of the trainer has been obtained and results, while not perfect, have been better than they might have been without the use of the trainer.

Case 1

Female, aged 6½ years. Left convergent strabismus; date of onset questionable. Refractive error: O.U. +1.50△ sph. Anomalous retinal correspondence, very unstable fusion, little amplitude, and no stereopsis. Visual acuity: O.D. 20/30; O.S. 20/40.

This child was spoiled, precocious, and very uncooperative. Treatment and occlusion were carried out with much difficulty. After fourteen orthoptic treatments her retinal correspondence was normal, her visual acuity was equal, and she had fair fusional amplitude with weak stereopsis. However, she did not transfer binocular single vision from the instrument to casual seeing for more than a half-hour period. After twenty-four lessons, the child was still not fusing on casual seeing, although she could fuse the Worth lights in the clinic. She was then given a television trainer, but she was so uncooperative that at first this could only be used for short periods at home. However, six months later she was using the trainer satisfactorily; fusional amplitude had increased, and she was fusing on casual seeing about 75 per cent of the time. After an additional three months, it was possible to give her bar reading exercises for practice at home. This youngster is still emotionally unstable. She occasionally has an alternating convergence, but she recognizes the resultant diplopia and fuses. I believe that without the aid of the trainer this patient would not have transferred her binocular skill to casual seeing.

Case 2

Female, aged 7 years. Right convergent strabismus following a fall at age 4. No fusion elicited. Refractive error: O.U. +1.25△ sph. Full optical correction with an add of +2.50△ sph. for near had been given. Recession and resection of muscles of the right eye had resulted in 30 diopters of divergence; present at the time of the first clinic visit.

After three months of orthoptic training, fusion with weak convergence had been established and the right medial rectus was resected. The patient still had a divergent squint of 20 to 25 diopters and could not fuse while using the television trainer. After considerable home training and six clinical visits, fair fusional amplitude with weak stereopsis was obtained. Six months after the second surgical procedure, a third was performed in which both lateral rectus muscles were recessed. This resulted in 15 diopters of *convergence.* The

television trainer was prescribed, and bar reading with the bifocal segment was begun. Three years after her initial visit, the patient was found to have enough casual fusion to maintain parallel visual axes about 85 per cent of the time. Bar reading is still difficult for her, and her fusional amplitude is still poor. I do not believe that this patient would continue to maintain parallel visual axes if the use of the television trainer was discontinued.

Case 3

Female, aged 4 years. Alternating convergent strabismus with right hypertropia. Age at onset of strabismus, 18 months. Optical correction for moderate hyperopia with considerable myopic astigmatism.

This patient came to the clinic frequently from 1950 to 1955. She was a most uncooperative and difficult child to treat until 1955, at which time she had her ninth birthday and became very conscious of her physical defect. She had been taught fusion on the major amblyoscope, but had made no effort to transfer the skill to casual seeing. She was given a trainer and has done so well with it at home that at the present time three consulting ophthalmologists are of the opinion that she is fusing at some distances and will continue to improve. She has had two surgical procedures and may require a third (her angle of deviation is +20 diopters at near), but the ophthalmologists feel that she will have good casual fusion by the time she reaches her teens. I am not quite so optimistic, but I believe that the cosmetic result will be much better because of the trainer, since she will not cooperate in any home training requiring parental supervision. This child controls the home, and many advantages are accorded her in the home because of her physical handicap. Therefore, she will not willingly relinquish this power, although she will make an effort on her own to maintain parallel visual axes when she is not in the home. She should soon be interested in the opposite sex, at which time it might be beneficial to resume her clinical training.

Case 4

Male, aged 5 years. No refractive error. Strabismus present at birth. Premature twin; twin brother, born last, has straight eyes. Visual acuity June 24, 1955: O.D. 20/30; O.S. 20/200. No fusion elicited. Anomalous retinal correspondence on Bielschowsky afterimage test.

Occlusion was prescribed for this child, but because of poor cooperation he did not achieve equal visual acuity until April 1956. When treatment was begun, his angle of deviation was 30 diopters of convergence, and by June 1956, after sixteen visits to the clinic, he had fusion on the instruments with fair amplitude and weak stereopsis. At this time a recession of the left medial rectus was performed, since the deviation had decreased to 10 diopters at distance, although there was still 30 diopters at near. After six postoperative visits the child was still not fusing casually, and when clinic visits were decreased, vision in the left eye was suppressed. His parents were very cooperative, and the child had become cooperative. Soon he was able to alternate freely and to fuse a light with red and green glasses at distances up to five feet. At that distance he could, with considerable effort, fuse on the television trainer, and this exercise has now been prescribed. It is too early to make a statement as to the efficacy of the trainer in this case, but I am hopeful. Here we have a cooperative parent and a child who enjoys television very much indeed and who, having decided to become a baseball player, is now desirous of obtaining good fusion. He is not much interested in the training devices at the clinic, although he enjoys coming to chat. At this point in his progress, I think that the trainer at home can be used to much better advantage than clinic visits, which can be decreased gradually and then resumed after he has had sufficient schooling for bar reading. I believe that his fusional amplitude will never become normal, because of the very early onset of his strabismus and the lack of normal retinal correspondence until now, at six years of age.

Case 5

Female, aged 7 years at first visit to clinic; now 10. Hyperopic correction: +4.00△ sph. O.U., with +0.50△ cyl. at axis 100 in O.S. Alternating convergence with no fusion. Vague history of onset of strabismus at eighteen months of age or earlier. Bilateral surgical correction had been attempted when the child was 2½ years old, and a second surgical procedure had been done when she was 3½.

Eleven months after her first clinic visit, this child had fusion with poor amplitude and weak stereopsis, but convergence was manifest about 75 per cent of the time. A television trainer was prescribed, and when she was examined six months later, she was found to have fairly good casual fusion. After another six months, in August of this year, she was found to have weak stereopsis, some improvement in amplitude of fusion, orthophoria with optical correction, a small angle of deviation without optical correction, and good casual fusion with occasional deviation at near with rapid recovery.

In conclusion, I wish to point out that not all of us have equal success with any given procedure, but my results with the television trainer have been most satisfactory in saving my time and the patient's expense and in encouraging the patient to strive for binocular single vision in casual everyday seeing. I believe that I am justified in my enthusiasm for this home training device as a supplement to clinic training, and I therefore urge that you investigate its possibilities for usefulness in your own practice. Finally, I would stress the importance of having the patient try this device at the clinic before you prescribe it for home use.

ANOMALOUS RETINAL CORRESPONDENCE: DIAGNOSTIC TESTS AND THERAPY

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ANOMALOUS retinal correspondence (ARC) still remains an important factor in the establishment of a cure of strabismus. In our experience, we find a large number of patients with strabismus and anomalous correspondence who achieve normal correspondence as a result of preoperative orthoptic treatment. We also find a few of these patients with persistent anomalies of sensory adaptation postoperatively; these may or may not develop normal correspondence as a result of postoperative orthoptic therapy.

Let us attempt to define normal retinal correspondence before discussing the abnormal type of retinal correspondence. Normal retinal correspondence exists when two images falling on corresponding areas of the two retinas have a common visual direction. In cases of strabismus, normal retinal correspondence is an indication of normal sensory localization of images despite a motor anomaly.

The age of the patient, his adaptability, and the intensity and duration of the anomaly are to be considered when treatment for ARC is undertaken. Fusion with normal correspondence and vergence control are indicative of postoperative binocularity of vision.[1]

ARC is manifested in two ways: (1) localization in two different directions of stimuli received normally by corresponding elements, such as the two foveas, and (2) localization in the same direction of stimuli received normally by noncorresponding retinal elements, such as the fovea of the fixating eye and an extrafoveal area of the deviating eye.

In normal eyes the images perceived are falling on the two foveas, so that fusion results. In strabismus the fixating eye receives the image on the fovea; the squinting eye, on a disparate or more peripheral area. Fusion is impossible, so diplopia results. Diplopia is not acceptable, so suppression at the fovea results, and the next step is the development of anomalous retinal correspondence.

So anomalous retinal correspondence becomes a means of adjustment or perhaps a close second to fusion. The eyes must have had past experience in binocular vision; otherwise diplopia could not be aroused.

Anomalous retinal correspondence is present in a high percentage of children with persistent tropia which had developed early in infancy. It is less common in children with tropia which had become manifest after the child was 3 years of age, or with small-angle deviations – 15 degrees or less.

Anomalous retinal correspondence may exist under various conditions. It may be present when the usually dominant eye is fixating but absent when the other eye is forced to fixate. It may be present in divergent squint when one eye is fixating and the other is diverging, but with binocular fixation a normal retinal correspondence may exist.[3]

Anomalous retinal correspondence is stimulated and strengthened by use and becomes unstable by disuse. This

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fact indicates the need for monocular occlusion.

There are certain groups of patients for whom preoperative orthoptic training is contraindicated: (1) patients with large deviations – 20 degrees or more; (2) young patients in whom anomalous retinal correspondence has not become established – those with recent onset of squint; (3) adults who desire only cosmetic correction.

Anomalous retinal correspondence is said to exist in a person when disparate elements of the two retinas, such as the fovea of one retina and an extrafoveal area of the other retina, have a common visual direction. Using corresponding retinal elements produces single vision; using disparate elements creates double vision or diplopia. In other words, a patient with anomalous retinal correspondence will see double when his visual axes are parallel and he is fixating with both foveas; he will see single when his visual axes are parallel but he is fixating with one fovea and an eccentric retinal element of the other eye. This can be demonstrated on the amblyoscope. At the objective angle, which is the patient's angle of deviation, where any redressive movement is no longer present, the patient will see double because he cannot superimpose the targets presented to each fovea. However, at a lesser angle the patient may see a single superimposed picture of the two targets presented, using the fovea of one eye and an eccentric retinal area of the other eye. A comparison of the objective angle, or relative position of the eyes, is made with the subjective, or localizing, angle to measure the angle of anomaly.

DIAGNOSIS

In a diagnostic examination, a patient with concomitant strabismus can be made aware of diplopia if the two fields are differentiated, as by a colored filter. If the colored filter is placed in front of one eye while the patient is fixating a small light, the patient will see a double image – one colored light and one white light. In esotropia the displacement of the images will be homonymous, and in exotropia, crossed, provided retinal correspondence is normal. Vertical displacement of the images can be seen in accordance with any vertical anomaly; the degree of displacement will coincide with the degree of deviation.

In patients with anomalous retinal correspondence, the localization will not agree with the position of the eyes. The subjective angle will be smaller than the objective, and the images may even be superimposed at zero; or the localization may be completely opposite. For example, a patient with esotropia may show crossed diplopia, and a patient with exotropia may have uncrossed diplopia.

If the objective angle (motor anomaly) and the subjective angle (sensory anomaly) are equal, the patient is said to have harmonious abnormal correspondence. If the subjective angle is less than the objective, the patient is said to have unharmonious abnormal retinal correspondence. If crossed localization occurs instead of uncrossed localization and vice versa, it is called paradoxical diplopia.

On the amblyoscope the same conditions may be found. If the patient superimposes the targets with the arms of the instrument set at his objective angle of squint, he has normal retinal correspondence; if he does so at a lesser angle, he has abnormal retinal correspondence. The abnormal retinal correspondence is harmonious if the objective angle is measurable and the subjective angle is zero; it is inharmonious if the subjective angle is less than zero.

The state of retinal correspondence can also be determined by the so-called afterimage test. This test utilizes a tu-

bular electric bulb with a band or ring which bears a fixation dot and also conceals a portion of the filament. The bulb can be turned either horizontally or vertically. With the filament lighted and the bulb horizontal, the patient's right eye is covered and the left eye fixates the dot for about ten seconds. The bulb is then turned vertically and the process is repeated with the left eye covered and the right eye fixating the dot. After the exposure, the patient will perceive an afterimage of two straight lines with a hole or gap in the middle. The position of the lines will indicate the visual directions of the two foveas. If the lines coincide to form a cross, and the two dots coincide, the patient has normal retinal correspondence. If the dots are displaced to the right or left, the patient has anomalous retinal correspondence and diplopia is present. The afterimage test is an indication of only the sensory relationship between the two eyes; a motor relationship also exists, since the position of the eyes is dissociated.

TREATMENT

Treatment of abnormal retinal correspondence is usually by three successive procedures: (1) occlusion, (2) major amblyoscope, (3) physiologic diplopia.

Abnormal retinal correspondence is a binocular condition; that is, both eyes are involved, but disparate retinal elements receive the impression of the object or the image viewed. The normal eye uses the fovea for fixation and has no area of suppression, but the squinting eye uses a peripheral element for fixation and has an area of suppression between the periphery and the fovea. Now, if fixation is switched to another area, the suppression area changes.

Occlusion. The logical first step in treatment is to occlude one eye constantly. It must be borne in mind that the angle of squint is not changed; that is, no motor change results, but the sensory pattern will be altered. Occlusion is alternated if alternating strabismus is present or if amblyopia is absent. If amblyopia is present, the better eye only is occluded until the vision in the two eyes becomes equal or within two lines of the Snellen chart. In this way, as vision improves, the sensory pattern can be altered. The age and occupation of the patient must be considered in the use of occlusion for psychological reasons.

Major amblyoscope. As soon as vision has improved, the major amblyoscope can be used, but only under the technician's supervision. With a Lyle major amblyoscope or a troposcope, we use alternate flashing at the patient's objective angle of squint, oscillation of the arms of the instrument and the monocular diplopia routine (also called the "Walraven technique," recently ably described by Frances Walraven, one of our colleagues).

The arms of the amblyoscope are set at the patient's objective angle of squint. We start with larger targets and progressively decrease them to fovea-sized targets. The lights are flashed alternately from one eye to the other. We increase the light intensity in front of the eye which has the lower acuity and is habitually nonfixating. The corneal reflexes are watched closely, as stimulus to accommodation may change the convergence angle, and foveal fixation may be lost. The foveas thus are constantly being stimulated by light. Some of the instruments have automatic flashing devices. I prefer to flash manually, since this provides less chance of my losing sight of corneal reflexes. The arms of the instrument are locked at the angle of squint, and a large side-to-side sweep is made within the angle of squint, while the technician again watches the corneal reflexes for any change due to accommodative effort. We also ask the pa-

tient to fix with the fovea of the squinting eye on a test target of foveal size while a larger target is gently oscillated in front of the squinting eye at an angle of about 20 degrees. This procedure is quite often referred to as "massage of the macula." The image of the target which is oscillating or massaging constantly invades the area of suppression in the anomalous region. The area of suppression becomes smaller, and the targets seem to be moving closer to each other; then the arc may be reduced. When the patient is able to superimpose the two images at his objective angle, the excursion is continued with alternate flashing again, until the pictures can be held fused for at least a count of fifty. This treatment is then followed by some amplitude training: holding fused images while symmetrical convergence and divergence are exercised. The technician utilizes the method which she feels is most suitable for the particular object in point. Some patients respond favorably in a short time; others take a longer time. We feel that after six, or even ten, sessions – rarely more than six – with decreased amblyopia, surgical intervention is indicated. Postoperatively we resume therapy, using the same techniques as preoperatively.[2]

The procedures of treatment which I have described are not necessarily applicable to all patients with anomalous retinal correspondence. There are certain patients who, despite almost perfect alignment of visual axes by surgical procedures, never gain a clear bifoveal fixation pattern. Eventually they again start suppressing, and they do not have binocular vision, even though preoperative treatment was given.

Patients must be "picked" for orthoptic therapy. Age, attention-giving ability, and attendance in the office are to be considered. Cooperation from parents and their understanding of what is desired are essential in selection of young patients for therapy. When a child is brought into the office for treatment of crossed eyes, and we institute exercise, patching, and glasses before a surgical operation, his parents must have some knowledge of what we are doing and why. Then, after the operation, the parents again may question, and rightfully so, what is to be gained by prolonged treatment. We must be prepared to inform them and to answer their queries reasonably, realizing our own limitations and not assuming the role of the ophthalmologist.

There must be a reason for the discrepancy between the patients who respond and those who never respond to treatment. Perhaps if a few of us will check our records, over a period of three to six years, for example, we can find something which we missed.

SUMMARY

Normal retinal correspondence in cases of strabismus represents bifoveal fixation at the patient's objective angle of squint. Abnormal retinal correspondence represents a perverted sensory adaptation to a motor anomaly.

Diagnostic tests of abnormal retinal correspondence, using colored filters, the afterimage test, and the major amblyoscope, are described.

Treatment by occlusion by the amblyoscope and by physiological diplopia are described.

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THE COLOR-DEPTH STRABISMUS TRAINING TECHNIQUE

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THE color-depth technique of training is one of the most recently developed techniques for training binocularity. It utilizes an overhead projector with screen, the correct-eye-scope, color-depth slides, and the stereomotivator (a slide disparator). Red and green goggles are worn by the patient. The correct-eye-scope is used for detailed near viewing, and the screen is usually used to stimulate peripheral and central fusion at greater distances, varying from about 35 cm. to 30 feet. The color-depth technique is unique in its wide scope of training possibilities. The following orthoptic procedures can be carried out by varying the targets and by altering the viewing conditions:

1. Antisuppression training on the correct-eye-scope can be carried out by using hand-and-eye coordination in the pointer technique. With one target, a dog is seen paramacularly with one eye and lines which reach diagonally from one border of the viewing area to the other are seen with the other eye. The patient who suppresses centrally at the angle of squint (which is compensated for by prisms or by putting the viewing device at the point of crossing of the visual axes) attempts to follow the lines with a pointer, as they cross from the periphery of the picture to the central area where they superimpose on the picture of the dog. To encourage diplopia and alternate fixation ability, the image before the dominant eye can be alternately flashed (by covering and uncovering the eye with the hand), while the suppressing eye fixes.

2. Fusion stimulation is similar to the red glass-muscle light recession exercise; i.e., superimposition occurs at the point of crossing of the visual axes and the patient is encouraged to maintain single binocular vision while the viewing distance is extended. Amplitude is encouraged by creating situations in which disparity of images is increased. This is done by mechanically producing base-out and base-in prismatic effects. Depth-perception appreciation and skill is thereby increased.

3. Accommodative squint can be treated by the above methods, and in addition, by the following techniques. Blurring of detailed targets can be required. Fusion with depth perception occurs when the eyes are straight; loss of fusion when the eyes diverge results in diplopia, but more easily noticeable is the loss of depth perception. Inquiries as to detailed aspects tend to stimulate accommodation. The gradual reduction in power of the lenses which permit single binocular vision can also be used as an approach to dissociation of convergence and accommodation. A near-viewing situation to stimulate accommodation can conveniently be produced by using the correct-eye-scope.

4. Often times, anomalous retinal correspondence will be present when the patient observes an object at one viewing distance, and normal retinal correspondence will be present when he views objects at another distance.

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This situation is occasionally found in patients with divergence excess who have intermittent exotropia at near and constant exotropia with anomalous retinal correspondence at distance. It is helpful to start treatment with the eyes in a position at which fusion with normal correspondence can be appreciated, since the tools for teaching correct visual habits are thereby present. The new stereomotivator permits training at different distances and therefore treatment can be adapted to the individual needs of each patient. Moreover, ideal situations need not be set up nor techniques used which are unnatural to everyday visual experience. However, it is not profitable to train patients who have abnormal retinal correspondence at all distances by this method (at least, this has proved to be true within the limits of our present experience).

5. Patients with exotropia or hypertropia can appreciate superimposition of images by the use of prisms attached to the goggles. The strength of the prism used is reduced as progress permits. Since usually peripheral fusion is stimulated first, and gradual progress is made toward foveal fusion, the problem of suppression is dealt with as it occurs, working from what is present and easily appreciated toward the more difficult. That is to say, even though there may be some suppression when superimposition occurs at the angle of deviation, the fact that the beginning targets are extremely peripheral usually allows the patient to develop some appreciation of binocularity and depth perception. Therefore, when these are not maintained, the patient usually tends to recognize diplopia rather than to suppress.

6. One of the principal advantages of the stereomotivator is in the treatment of patients with divergence excess or divergence insufficiency in whom natural distance viewing can be stimulated.

7. In addition to its other uses, the stereomotivator is exceptionally reliable in the determination of the presence or the absence of stereopsis, even when a large angle of squint is present because of peripheral stimulation which it produces. It is, therefore, valuable in evaluating progress in binocularity.

The color depth method of training binocularity utilizes the following favorable techniques:

1. Patient participation is required. Approaching the picture stimulates convergence, and recession stimulates divergence; in addition, fusional amplitude is developed by the mechanically produced base-out and base-in prismatic effect. Also, hand-and-eye coordination requires the patient's attention.

2. Training can be carried out at all distances. This is of special value in cases of divergence excess and of divergence insufficiency.

3. Peripheral or central vision can be stimulated by changing the slides and/or viewing distance.

4. The use of instruments for convergence is eliminated.

5. Several patients can be trained at the same time.

6. Natural viewing conditions are maintained, and stimulation of the most natural visual skill, viz., stereopsis, occurs.

7. The method has high interest value. Motion picture or television viewing is invariably accepted enthusiastically. Constant variability of degrees of depth is a challenge to the patient's power of perception and stimulates his concentration.

The stereomotivator is a notable addition to orthoptic training methods, and its wide range of stimulating uses makes it a valuable addition to any orthoptic office or clinic.

ALTERNATING ESOTROPIA

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THE case which I am going to present is one of completely alternating esotropia, having no superimposition at all, having existed for over ten years, in a boy, age 16, and in which complete fusion was obtained. This was demonstrated in all tests for depth perception, as well as more than ordinary ability to converge, diverge, and overcome prism base-up and base-down. This case is selected for presentation because generally the orthoptist hesitates to give the ophthalmologist much encouragement on this type of problem. An occasional record of recovery from a seemingly impossible situation like this may stimulate further work along this line.

CASE HISTORY

Kenneth had had an alternating esotropia since he was 5 years old. When he was about 5 years old, he fell off a porch with a knife in his hand. According to his parents, the knife penetrated his skull, over the left brow. He was unconscious for about twenty-four hours. About one week after this accident his right eye began to turn in. Just prior to his first visit to our office he began complaining of headache, nausea, vomiting, and pains in the right eye after doing close work. His general health was good. There was no history of eye trouble in the family which would have a bearing on his case. He was not a nervous individual and appeared to be normal in all respects. Prior to our examination he had been fitted with corrective lenses, but he had not worn them for quite some time.

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Refractive and Orthoptic Examination

Examination showed him to have 20/15 vision in each eye, and an esotropia of 40△ for near and for distance. By retinoscopic examination under cycloplegia, he was found to have a moderate degree of farsightedness in each eye: O.D., +1.75 sph., −25 cyl. axis 180 and O.S., +1.50 sph., which equaled 20/15-2 in each eye. With this correction in both eyes his vision was 20/15. The fundi and media were negative.

On the troposcope he had an objective angle of 50△ and questionable momentary fusion with oscillation. On the cover test for both near and distance he had 40△ of esotropia. His near point of accommodation without glasses was 62 mm. for the right eye and 68 mm. for the left eye; it was 50 mm. with both eyes. On the Worth 4-Dot test he saw either two or three lights. His ductions and versions were normal.

Glasses were ordered and arrangements were made for him to start orthoptic exercises to be given four times a week. After sufficient exercises to give him diplopia he was to have a surgical operation to straighten his eyes, followed by exercises to develop fusion and coordination. The prognosis was for eventually straight eyes with third-degree fusion. This was in September 1952.

Kenneth never came in for his exercises, and it was learned that the family had consulted another doctor who told them that the whole idea was foolish. As a result of this, the course of treatment was cancelled. However, due to

the fact that the Crippled Children's Society, service clubs, and the school nurse were very much interested in Kenneth, the family was not allowed to drop the case. Members of these organizations were aware of the boy's intelligence, conscientiousness, athletic ability and pride. Through their efforts the parents were persuaded to bring Kenneth to the office for a new start, with the understanding that they would follow through on the treatment as outlined by the doctor.

In October 1953, a little over a year after his first visit, Kenneth was again given an examination. The findings at this time were essentially the same as at the original examination, except that he had become a little more farsighted.

After the examination the plan of treatment again was outlined for the parents – orthoptic exercises, surgical correction, and more exercises. They were told that unless we could be assured of complete cooperation from them and Kenneth, we would not be interested in taking his case for treatment. Inasmuch as one of the interested organizations was paying for his treatment, we felt that it was essential to have a complete understanding with the family. Often when the financial responsibility is taken on by a third party, medical treatment is taken too lightly. The family agreed to cooperate one hundred per cent.

Two days later Kenneth was started on his first series of exercises. Our exercises are given in courses of twelve exercises each, and at the end of each course the patient's progress is checked by the doctor. He was given thirty minutes of exercises, five days a week.

Treatment

Kenneth's treatment consisted of alternate occlusion and vigorous fusion stimulation on the troposcope.

At the time of his first exercise his objective angle was 60△ esotropia on the troposcope. We worked with him at this angle to develop superimposition. His concentration was excellent. After his third exercise he could superimpose momentarily off and on during the exercise. On the fourth exercise he had correct diplopia at zero on the troposcope. As the days went by, he was able to superimpose for a longer period of time. At the end of the first course of twelve exercises he could alternate fixation at will and hold the superimposed targets with oscillation. At the end of three weeks he could superimpose without oscillation. We also learned at that time that he had not been wearing his glasses all the time even though he had been instructed to do so. The doctor re-emphasized the importance of wearing the glasses. To aid him in getting started with the constant wearing of them, atropine was instilled in each eye.

During the second course of exercises Kenneth developed grade-2 fusion, and the angle of deviation was steadily being decreased. "In-and-out" and "chasing" were started, combined with the flashing of both eyes. He was started on physiological diplopia exercises at home.

The third and fourth courses of exercises produced steady improvement. At the end of the fourth course, which was approximately one and a half months after his first exercise, his angle of deviation had been decreased from 60△ to 43△ and his fusion was holding fairly stable with some degree of amplitude. He was wearing a patch constantly, due to the diplopia which he had worked so hard to induce as a prerequisite for surgical intervention.

The operation was performed on February 10. A 3-mm. recession of each medial rectus was done. On February 15 Kenneth returned to the office. He

was found to have 3△ esophoria for near and no esophoria for distance. There was no excessive inflammation, and an exercise was given the same day. The subjective angle was +10△, the objective angle was +7△ with divergence to −7△. He fused at +5△. Two days later he returned to the office, and the sutures were removed. With his optical correction in Green's refractor and using the red glass, his angle measured 9△ base-out and 2△ base-up in the left eye at 20 feet. Using the white dot his amplitude of fusion was 12△. He was able to fuse at 12△ base-out and 2△ base-up on the left eye; his amplitude of fusion was about the same. Without the base-up prism on the left eye and with a slight head tilt he could fuse satisfactorily.

During the third exercise after his operation, his subjective angle was still +10△ and he was fusing at that angle also. His amplitude of fusion was 20△. On the fourth exercise his subjective angle had increased to +18△ and his amplitude of fusion was decreased. In general, it was a little bit discouraging. We started using 0.5 per cent atropine, urged him to avoid straining to see any close work, and gave him 5.5△ base-out clip-on prisms to wear. At this point his exercises were increased to one hour each day in the office. At the end of his first course of treatments after surgical correction, his subjective angle was reduced to +12△; he could fuse at +9△ and diverge to −20△. During the second course, we worked very hard on fusion training, trying to reduce the angle. On the last exercise of the course, with his glasses, the subjective angle was reduced to +3△ and he could fuse at zero with 10△ of divergence. On his twenty-fifth exercise after surgery, the clip-on prism was removed completely. The subjective angle was +10△ and he could fuse at +5△. He could diverge to −15△. A week later he reported that when he viewed motion pictures he had single vision all the time and that sometimes on looking closely at an object he saw only one. Two days later he reported single vision all the time. In just a few days after that his subjective angle was reduced to +3△ and he was fusing at zero with a 32△ range of amplitude.

Kenneth was getting pretty tired of exercises at this point, so we gave him a three-week vacation. During his vacation, however, he was to work very hard on push-ups. He was told that if he continued to improve, we would give him an orthofuser to use at home and his exercises would be cut down to one a week.

When we tested him at the end of three weeks, his eyes were staying straight all the time. His vision with glasses was 20/25+2, O.U.; without glasses, 20/15−1, O.U. At this time he was started on home work with the orthofuser, push-ups, jump convergence, and physiological diplopia exercises, and told to return in one month.

At the end of the month he was checked and he had held his own. He was allowed to discontinue wearing glasses at this time and to come in only once a week for an exercise. Without his glasses his angle of squint stayed about the same and his amplitude increased. His measurements were the same for near and distance, +5△ esophoria. We continued to stimulate his fusion and increase his amplitude. He was also given the base-in orthofuser to use at home along with the push-ups and other home exercises he was doing.

On October 19, 1954, almost a year after his treatment was first started, his regular exercises in the office were discontinued, and he was instructed to keep up with the home exercises. On that date his versions were normal in all directions, his subjective angle was +7△; and he had fusion at zero and an amplitude of 50△.

On January 25, 1955, he was in the office again to tell us that he was leaving for another state to work and would stop in the first time he was home for a visit. His measurements on the troposcope then were subjective angle, +12△; fusion at +5△; and amplitude of 55△. His versions were still normal. His near point of convergence was 60 mm. As far as we know he is still getting along fine, and we are anxiously awaiting his visit home so that we may again examine him.

This case was one which just had to turn out with excellent results. You will remember that I mentioned earlier that there were several organizations much interested in seeing that this boy should receive the maximum care and treatment for his eye condition. One might say that the spotlight was on us, and we knew that his case had to be corrected. Realizing that the parents were doubtful of the outcome, we were not certain that all instructions would be carried out even though they had promised to do their part. It was with gratification that we were able to see this case through.

READING DISABILITIES

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In considering the reading problems of children, the purpose of reading was discussed.

First, we read for information; second, for pleasure and vicarious experience – escaping the bounds of reality.

Ten to fifteen per cent of school children have difficulty in learning to read. Since many school subjects depend upon reading, inability to read is an important cause of school failure. This, in turn, seriously undermines the child's self-respect and self-confidence, which may jeopardize not only his academic achievement but his personality development as well.

Early recognition of reading disabilities and prompt institution of appropriate preventive and remedial measures by the school is necessary. With early treatment by well-trained teachers and psychologists, the prognosis is good. The longer the problem is neglected, the more difficult it is to remedy, because of the adverse emotional attitudes which frustration and discouragement have deeply implanted within the child.

The possible causes for reading failure are numerous. One must first ascertain that the difficulty is not simply a symptom of general retardation rather than a specific disability. Chronological age is no indication of reading readiness. A *mental* maturity of about six years is necessary for reading ability. However, many children do not attain this mental age until sometime after their sixth birthday and their entrance into first grade.

Speech facility and vocabulary development were discussed as other factors in reading readiness.

Emotional and social development were also considered.

Children who are so shy and retiring or so aggressive and hostile that they cannot get along with their classmates, or those who are so restless and distractable that they can't concentrate on their studies, are most likely to obtain a poor start in reading. Pressure exerted to make them read when they cannot conditions them emotionally against reading. They become afraid of failure, resentful toward the teacher, or unwilling even to try to read. Such attitudes toward reading may persist long after the child has acquired sufficient maturity to learn to read, and if uncorrected, they may result in continued lack of interest, poor cooperation and failure. Therefore, in cases of mental and emotional immaturity, reading should be postponed or at least not pushed until observation and tests prove that the child is ready for it.

Reading also depends upon adequate vision and hearing, as we well know. Defective vision, refractive errors, eye muscle imbalances, and poor coordination of the two eyes should always be looked for in nonreaders. Some people feel that an audiometric examination should be made as a matter of routine.

Chronic illness or fatigue, resulting in poorly sustained attention and application in school, and frequent or pro-

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longed absences from school because of illness may prevent satisfactory progress in reading.

Prolonged emotional disturbance, severe social maladjustment, and an undesirable emotional relationship with the teacher may also interfere with adequate application and effort in reading as well as in other school subjects.

The advantages and disadvantages of the whole-word, or "sight," method, and of the phonic method of teaching reading were discussed, and a combination of the two methods was favored.

The first step in treatment of reading disabilities is to assure the child that his difficulty is no reflection on his intelligence and to make every effort to build up his self-confidence, willingness to cooperate, and desire to read. Not only must the reading materials be at his own level of ability but also they must be suited to his level of interests.

The overanxious parent is probably more harmful than helpful in teaching the poor reader, and aid should be sought outside of the home from competent people specially trained in this field.

Treatment of a reading disability should begin with a complete medical and psychological examination of the child.

Emotional factors, more than any other one cause, have been found to be primary in reading difficulties. Teachers of remedial reading should be specially trained in the observation of emotionally disturbed children and should work in close cooperation with a psychologist.

Typical and difficult cases of reading problems were discussed.

The psychosomatic aspects of visual disturbances in the light of psyschoanalytic theory were discussed, following the ideas of Weiss and English in "Psychosomatic Medicine."

The ophthalmologist is in a position to observe many symptoms due to emotional conflict. Visual experiences which have been traumatic to the individual may result in squinting, watering of the eyes, hysterical blindness, reading difficulties and other symptoms.

A careful review of the literature indicates that ophthalmological medicine is not keeping abreast of other medical specialties in psychosomatic research. More critical research is indicated in this vital field.

Certain types of refractive errors seem to have little to do with reading difficulties. Some studies show that whereas children with hyperopia showed smaller reading gains in comparison with reading gains of a group of children with normal vision, those with myopia showed greater reading gains than did the normal group.

Eye muscle imbalance appeared to be significant in reading difficulties. The serious effects of muscle imbalance upon reading indicate the need for careful examination by a competent ophthalmologist. Since orthoptic training materially alleviates ocular muscle weaknesses, this training should be an early therapeutic procedure in cases of reading disability in which this eye muscle imbalance is present.

Alexia, or congenital word blindness, was discussed and considered to be an extremely rare, if not mythical, concept.

It is not a useful diagnosis, and in cases so diagnosed, the patients have been found to respond satisfactorily to variations, sometimes of a radical nature, of psychological or educational techniques.

In conclusion, the multidiscipline approach was stressed in the solution of reading problems.

Real cooperation is necessary between the ophthalmologist, orthoptist and psy-

chologist. The diagnosis of a psychogenic basis for a reading or visual defect should be followed up by referral to a psychologist for diagnostic evaluation.

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ADULT ORTHOPTICS

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The scope of this title is obviously limitless and suggestive of extensive treatment beyond the possibility of a paper of this nature. The two words are somewhat ambiguous; they have several meanings, depending on their significance to each individual hearing them. Do they mean orthoptics performed in an adult manner on adults? In this instance, I choose this inference.

To practice adult orthoptics in an adult manner requires no little skill of the orthoptic technician. It is a work demanding much patience, great tact and more than a superficial knowledge of orthoptics and of human psychology. Much of our training and practice is in the care of children's cases, and often a technician who does some self-searching finds that he or she is not as well versed in the psychology of adults as it would be comfortable for him to be! In my observation of other technicians at work with adults I have noted much variation. Not every technician seems adept at it, although some do wonderfully good work in this respect. Some of the procedures that these technicians have evolved are very effective, yet there is not one of them but would deny that his method is the last word. They, as you and I, know there is no place for unquestioned routine or complacent following of unvaried techniques in the attainment of a successful orthoptic cure. Living as close as we do to our work, we must not become insensitive to constructive criticism or suggestion lest we allow the very dynamic work we do to become crystallized and, in consequence, less effective. It is my thought that we orthoptic technicians, probably more than many other people who work with adults, need careful training in psychology, and when it has not been a part of our training or educational background, we must remedy this lack through extention work with some college or university or through our own reading.

Adults need a valid motivation, even as do children, for making the effort required of them in the treatment of their particular muscle problem. Children frequently do what is asked of them in orthoptic treatment without obvious resistance, and they do it largely because they wish to please us or their parents. The adult patient, however, comes into the orthoptist's office with a great variety of attitudes. Sometimes he is obeying an ophthalmologist complaisantly while utterly ignorant of why he is seeing the orthoptist. He *may* remember that his doctor said something about a muscle imbalance or that he was to have exercises, but his ideas about it are vague. Then there is the other extreme – the patient who comes in with his prescription memorized or readily at hand on a bit of note paper along with other bits of pertinent information, often including a list of subjective symptoms. In between these types is the patient with a little education on the subject of visual difficulties. He is curious about why his eyes are so uncomfortable, but his interest in getting relief may fluctuate. For this patient we orthoptists must supply motivation for treatment and do it through educational methods appropriate to his case.

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A major responsibility, this education of the patient – and it seems to me to fall squarely on the shoulders of the orthoptic technician. Without knowledge of why he should submit to orthoptics, the average adult will have none of it; he often expresses this vocally but more often simply by not showing up for the first treatment. This seems to me a great waste of his ophthalmologist's time, of his own time and opportunity, and of our time as well. Since successful treatment depends so largely upon the patient himself, it is apparent that he needs some explanation meaningful to him of what we can do to help him help himself. Time spent in getting the patient's acceptance of orthoptics as an important ophthalmic service is time well spent. The almost universal idea that lenses are a cure-all and that 20/20 vision is perfect sight often makes the patient somewhat skeptical on being confronted with orthoptics for the first time.

So it seems that the first step we must take to get our adult patient to accept treatment is to recognize that we are working with a human being and that it is his very humanness that often creates some of his visual problems. Some adults are difficult to work with, and we might be forgiven if we are looking for a "model patient," but we must put ourselves on the adult plane of life and remember that the model patient is the one who gets a cure!

Assuming that the patient is in the examining room and that you have reminded yourself, however grudgingly in some cases, that he *is* a human being, you will have in mind that he will be subject to all manner of diseases, ailments, and weaknesses, and will have many a symptom not neuromuscular! You will further remember that, even as you and I, he is plagued or blessed by emotions. He may be anxious, inhibited, withdrawn, overly aggressive, or, as the majority of us, subject to feelings of inferiority. With these or similar thoughts in mind proceed with your history taking. Let the patient talk while you listen – really listen. Often this is the most important moment for the patient, and it can be for us for obvious reasons. Talking often serves as a safety valve for worried or perplexed people to relieve inner pressures; and incidentally, it makes your patient feel more at ease, more receptive to you. Of course, some patients will turn on an almost ceaseless flow of seemingly irrelevant chatter, but curb it only occasionally at this important first meeting. You can get the patient back to the real clues by your questions when you feel that he needs to be brought back to them. Usually the patient will need to be drawn out, because he feels that his symptoms are so vague as to be unimportant or that his discomfort is just one of the things in life that has to be borne. Sometimes you will find that your attempt to get a little history of symptoms and their onset will be futile, because of the patient's reticence or embarrassment, or because he lacks the means to express his trouble. In such a case, you have doubtlessly found that in getting on with the examination, pertinent items of history will be forthcoming. Very often the patient feels grateful to you for listening well and understandingly to him, and this can form a basis of his determination to cooperate with you.

One important phase of the history taking is learning what kind of work the patient does – what his visual tasks are – for often this is of crucial importance to us as we proceed with his rehabilitation. Besides the types of visual tasks performed, information about the patient's health should be learned. The symptoms of heterotropia and heterophoria cannot determine a diagnosis

unless the presence of such conditions as anemia, malfunction of the thyroid gland, and allergic asthma has been investigated. Often this information is welcomed by the ophthalmologist, since orthoptic failures occasionally can be traced to such systemic conditions. The doctor can then ask the patient to have a thorough physical examination, followed by whatever medication or treatment is indicated.

When I have as much of the patient's history as I think I am likely to get at this first session I proceed in much the same manner as I have seen practiced in other orthoptic offices, determining his visual acuity both with and without glasses for distance and for near point. This I follow with the Worth lights test, prism cover test, and in most cases of heterophoria, the phorometer test as well as the Maddox rod test. Some of these tests have been performed previously in the ophthalmologist's office, but I repeat them because the results sometimes differ, often because of the emotional state of the patient. Then I observe the rotations of the eyes and make such measurements as are suggested by the comitancy or incomitancy noted. In cases of heterophoria I usually do diplopia tests, using a light red filter. I do this first with the filter over the right eye, then with the filter over the left. This provides dramatic evidence of fusion failure. This test is customarily done in cases of heterotropia as evidence of primary and secondary deviations, but I feel that it serves a good psychological purpose in cases of phoria, particularly when the patient learns that he is seeing double in one or more positions of the eyes. Many times I precede the diplopia test by the Prince rule test for accommodation; sometimes I reverse the order of the two tests, for no particular reason. Next I measure the near point of convergence, both objectively and subjectively. I note the eye that gives up fusion first, as this is often conclusive evidence of which is the dominant eye when there has been some doubt up to this point. After all these tests, when the patient has a phoria I use the telebinocular for the following tests: phoria test at distance and at near point, vertical phoria test at distance and at near point, and then the Keystone fusion test at both distance and near point. These tests are simple but convincing evidence to the patient of certain phases of his visual upset, and for us they serve as correlatives to tests already performed. The Keystone stereopsis test provides good evidence also, since most people find the loss of depth perception quite revealing. Often during these tests or after they are performed, the patient questions what is happening; at this time it is well to spend a few minutes clarifying such terms as fusion, accommodation, suppression, and stereopsis, and to answer particularly any question the patient may advance. Often a patient will exclaim on being apprised of his performance on the depth-perception test, "So that's why I have trouble parking my car!" or some other equally revealing remark. Now I place the patient at the major amblyoscope (troposcope) and proceed with the measurement of the angle of deviation, fusion or quality of fusion, fusional convergence, divergence, recovery, and suppression, or with any measurements the type of case seems to demand. When the patient has a phoria, the suppression phenomenon usually evokes questions which should be dealt with, since it has an important part in the looked-for cure; if no question is forthcoming, it is well to demonstrate its importance to the patient.

Now the patient often feels that all that remains to be done is to have the appointments set for treatment! With the information gleaned from the foregoing tests the orthoptist can make at

least a tentative diagnosis, if not a final one, and plan the treatment.

Nothing in the foregoing remarks has been new to you, I am certain, except perhaps the suggestion of the value to the orthoptic technician of knowledge of psychology.

Although we usually do not select our patients for treatment, a large part of our job is establishing the prognosis. For this we need every bit of understanding of the basic types of strabismus and of the physiologic and psychologic needs of each patient we see in order that we may make as intelligent and constructive suggestions to the ophthalmologist as possible. Without knowledge of psychology we cannot be effective orthoptists.

With more knowledge of how the mind and body affect each other in sickness and in health we will be able to practice a more adult type of orthoptics.

At this point I want to ask some questions. If a patient has consulted his general physician, then his ophthalmologist, and then has been referred to us, and after as careful examinations as we can do and with what seems sufficient orthoptic treatment there is still no relief of symptoms, what then? Often, the patient is re-examined, sometimes more than once, by his ophthalmologist, and many times he is referred to us again. We make another effort, still with no relief to the patient. Do we then give up? Do we put the patient into the too convenient category of "neurotic" and let him go back into the stream of life no better off or perhaps worse because of the fact that so many of us have not helped him? It is true that these cases are few, but they stand out from the mass of orthoptic cases as unpleasant monuments to our collective failure.

Would the adult procedure be to refer him to a psychiatrist or to the Family Counseling Service or some similar community agency where there is expert social psychiatric help available at minimum cost to the patient? Some of the ophthalmologists with whom I work think so.

We all come to know our patients quite well since we often see them a number of times. In the course of this association they tell us of events and conditions in their lives, seemingly not directly part of their visual problems but suggestive of emotional states that could be linked to the eye trouble. If we are looking to a cure and we suspect that the emotional factor in a patient's case is considerable, should we ignore it? Should we discuss this with the ophthalmologist? Should he talk with the patient and suggest that he avail himself of the help of appropriate agencies? Do we need to feel any concern about the mental health of our patients?

SUMMARY

I want to make clear a point which I may not have emphasized sufficiently. My principal point is that we orthoptic technicians need education in psychology. It should be included in our orthoptic curriculum in order that we may make a more adult approach to solving the problems that are entrusted to us by ophthalmologists. We need to recognize that mankind has difficulty in adjusting to our rapidly changing environment emotionally and physically. In assuming this adult approach we should consider the help that the various psychiatric social agencies offer perplexed humanity. And last, but certainly not least, I am not saying that we technicians, or for that matter the ophthalmologists, should attempt to psychoanalyze our patients – only that we should become sensitive to these problems and aware that this facet of our work exists.

SURGERY FOR ESOPHORIA IN THE ADULT

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FEW ophthalmologists consider surgical intervention in the treatment of phorias, yet it is my conviction that many more patients with phorias can be helped by such intervention than is commonly realized. If a diagnosis of a muscle anomaly can be made in a case of phoria, the results of treatment are more satisfactory when the anomaly is corrected before binocular vision is lost and tropia is allowed to progress. However, a patient with phoria, but with no disturbing symptoms should not be subjected to surgical treatment.

It has been my experience that the phorias require as extensive, if not more extensive, surgical correction than do the tropias; and the prognosis is better in the phorias because the patients already have some fusion. Orthoptic training often alleviates the symptoms of esophoria but seldom decreases the amount.

In reviewing the voluminous literature on eye muscle conditions, I discovered that surgical treatment of esophoria has received a minimum of attention. The only article that I found on this subject was by Stine, in the September 1951 issue of the American Journal of Ophthalmology. He reported 15 cases; he recessed one medial rectus in 14 cases and both medial recti in the other case. In 10 cases he found what he considered to be muscle anomalies.

In reviewing my cases in which surgical procedures on eye muscles of adults had been performed, I found 14 cases of esophoria in which I had resorted to surgery in an effort to help the patient. In 12 of these I did a recession of one medial rectus, and in 2, a bilateral recession of the medial recti. Definite improvement resulted to a greater or lesser degree in all cases, and in only one case did I have any postoperative worry. In the latter case, the patient was a premedical college student who had been examined by me and by other ophthalmologists over a period of years because of his complaints due to esophoria. He reached a point at which he could no longer study, and unless he could obtain relief, he planned to drop out of school. A bilateral recession of the medial recti was done, and for a few days he had a very disturbing diplopia. This cleared suddenly, and since that time he has been symptom-free and his eyes are no longer a problem to him.

A detailed history will not be given of each case, but there are certain facts which stand out in a review of this admittedly small series. Scobee pointed out that, for some reason or other, patients with esophoria are almost always intelligent and "high-strung." These patients of mine certainly fitted into that category. They included three high-school teachers, one college teacher, two bookkeepers, two college students, an attorney, and a physician. Most of these individuals had consulted two or more ophthalmologists and had been given prisms, bifocals, orthoptics, and, in two cases, even psychotherapy. Three had intermittent diplopia, especially toward the end of the day. Most of these patients had been

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observed for more than two years and several, for five or six years before it was decided to try to help them surgically. I feel that these patients were almost universally grateful for the relief which they received.

The objective improvement in these cases was not as great as I would have liked; in most of them I feel that I made too small a correction, for esophoria in some degree persisted. Yet the subjective improvement was marked. Possibly some of the improvement was psychological, but all of the patients were able to return to their studies or their work and to carry on without the serious disabling symptoms which they had had before the operation.

In summary, then, I should like to state that too few adults with symptom-producing esophoria are given the benefit of surgical treatment.

OPTICAL AIDS TO ORTHOPTICS

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FOR many years the importance of refractive error in relation to heterophoria and heterotropia has been well recognized. Consequently, patients now referred to the orthoptist are wearing the lenses that provide for the best possible visual acuity and allow for the muscular imbalance present. Thus, the patient with esotropia wears the maximum hypermetropic and minimum myopic correction, and the patient with exotropia wears the maximum myopic and minimum, if any, hypermetropic correction consistent with good visual acuity. I should like to review the additional optical aids which the orthoptist can use to treat these conditions.

Bearing in mind that the one aim and whole purpose of orthoptic treatment is the comfortable maintenance of binocular single vision, accessory optical aids which do not help should be discontinued after a fair trial. So before use of such optical aids can be considered, all other efforts to obtain binocularity should have been made. The patient should have good simultaneous perception and fusion on the troposcope or synoptophore, with good amplitudes and negligible suppression.

The advantages obtained from the increase or decrease of a spherical correction are solely dependent on the accommodation-convergence relationship. The normal occipital accommodation reflex results in the exertion of closely corresponding amounts of accommodation and convergence. Thus, a blurred image stimulating accommodation induces convergence also; and diplopia, stimulating convergence, results in accommodation too. This is an ancient inborn reflex; it is not controlled by the will. However, discrepancies between accommodation and convergence are adjusted by the harmonic accommodation reflex, thus making the fixed relationship a very elastic one. This again cannot be initiated voluntarily; it develops only with usage and appreciation of the reward of the clear binocular image obtained. However, the frontal accommodation reflex enables a voluntary effort to be added to accommodation and convergence.

The increase in strength of convex "plus" lenses reduces the amount of accommodation and, hence, of convergence. Thus in cases of slight esotropia, even with the prescribed correction, this may well result in binocular single vision. This occurs especially in postoperative residual esotropia when the deviation is most elastic – hence the importance of immediate postoperative orthoptic treatment. The use of atropine for this purpose has the disadvantage that accommodation does not occur at all; furthermore, when the drug is stopped, accommodation is very poor for ten to fourteen days. The increase in the "plus" correction may only be necessary for near vision, as in the many cases of intermittent esotropia in which there is binocular single vision for distance and manifest esotropia at near. In these cases, bifocals have been found to enable binocularity to be obtained at near too. The most effective lens has been found to be the "executive" type, or full half-segment. The

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most economical form of lenses for treatment is an additional segment to be cemented on the patient's glasses, which eliminates the necessity for new lenses and facilitates replacement with a segment of reduced strength when required or complete removal of the segment. The difficulties sometimes experienced by adults when first wearing bifocals do not seem to be noticed by children who are treated with them. A sure indication of their effectivity can be appreciated when parents state that the child raises his chin to use the lower segment, thus showing appreciation of the binocularity obtained. I have not followed enough of these cases to establish whether or not comfortable binocular single vision is permanently established after the bifocal is discarded.

A decrease in power of the convex lens stimulates accommodation and hence, convergence and may well result in binocular single vision in cases of exotropia, especially exotropia following surgical treatment for esotropia. This situation is rarely seen but does occur, and the addition of "minus" lenses invariably reduces the deviation and permits fusion. In cases of intermittent exotropia, with exotropia occurring for distance and exophoria, for the most part, at near, clinically "minus" lenses result in a greater range of binocularity, but the advantage of constant wearing of these lenses, I have not had the experience to observe.

Temporary accessory minus lenses can be of help in the treatment of accommodative convergent strabismus in cases in which the prognosis indicates that binocular single vision can be obtained and glasses discarded. In these cases of esophoria with glasses and esotropia without glasses, this is possible when the refractive error is +3.00 D. sph. or less. Treatment consists of elimination of suppression so that diplopia is well appreciated when the eye deviates. "Control" is then taught by relaxing accommodation to fuse the two images. This step can usually be quickly learned, even by a very young child, but the disassociation between accommodation and convergence is much more difficult and is not possible until the patient is 7 or 8 years of age. This is, in fact, developing the harmonic reflex; accommodation has to be exerted without convergence and has to be learned and firmly established. At this stage of the treatment, a gradual decrease of the correction by additional minus lenses of increasing strength, starting with a –0.50 D. sph., or even a –0.25 D. sph., is invaluable, and the most important evidences of the effectiveness of this method are bar-reading ability and the presence of binocular visual acuity.

The most effective method of increasing or decreasing any correction is the use of additional clip-on lenses which leave any astigmatic correction undisturbed and only temporarily change the prescription. Since I have been practicing in the United States, I have neither used nor seen "clip-ons" such as I have just described. Perhaps this can be attributed to the variety in shape and size of the glasses worn by the children here, which would necessitate individual clip-ons being made. I used to have frames with standard interpupillary distances and sets of interchangeable lenses to fit each frame. Thus clip-ons of any strength could easily be loaned to any patient.

The strength of the accessory lenses can be decided by trial and error. It can easily be noted whether these lenses decrease the deviation; often there is no change. If binocular single vision can be obtained at any point, the additional lenses should be worn constantly; a mere reduction in the deviation without binocularity is not enough to warrant the wearing of clip-ons. Vis-

ual acuity with the extra lenses should be estimated. In the case of additional "plus" lenses, slight blurring for distance can often be tolerated; this blurring, however, in a case of esotropia with a strong accommodative, especially a hyperaccommodative, element may result in an excessive attempt at accommodation and hence a convergent spasm. Both the parent and the child should realize that the extra lenses are not for visual improvement. The period of time the lenses are worn depends on the development of binocularity. As soon as the cover test shows a good binocular recovery for distance and at near, and fusion is demonstrated with the Worth dot test for distance and at near, the strength of the lenses may gradually be reduced (often only by 0.25 or 0.50 D. sph.), until they are finally eliminated. This period may vary from four weeks to six months. However, it must be remembered that wearing lenses that result in an overcorrection of hypermetropia for long periods results in a convergence deficiency.

During treatment in the orthoptic clinic the "plus" lenses can help the patient with esophoria to obtain divergence (binocular abduction) on the troposcope or the synoptophore. The patient with fully accommodative convergent strabismus who is attempting binocular single vision without glasses, at the expense of clear visual acuity, often finds the exercise difficult; the momentary addition of a +5.00 D. sph. for each eye often helps him.

"Minus" lenses on the troposcope greatly aid convergence and there are many patients with convergence insufficiency who are unable to converge without their aid. It seems that once good convergence is established with additional —4.00 D. sph. for each eye, normal adduction can easily be obtained. Too, the troposcope is usually used to test distance vision; with an additional —4.00 D. sph. it can be used to test for near. The normal relationship of accommodation and convergence being one diopter of accommodation to one meter angle of convergence, the extra 4 diopters of accommodation results in the normal equivalent of 16 prism diopters (8 degrees) of convergence; the amount varies but it should be taken into account. The value of the near test on the troposcope can be demonstrated in the diagnosis of the state of binocular vision in the case of exotropia. When simultaneous perception and fusion are not elicited normally, but are found to be present with —4.00 D. sph. (as is often the case), the prognosis is greatly improved. Recently, cases of variable esotropia have been tested with glasses and —4.00 D. sph. on the troposcope. In the treatment of these cases the first aim is the elimination of all suppression. There are two areas of suppression in the squinting eye: suppression of the macula to avoid confusion, and suppression of the area of the retina receiving the same stimulus as the macula of the fixing eye to avoid diplopia. It is important that suppression in both these areas be eliminated, for without first appreciating diplopia when squinting, the patient cannot attempt full binocular single vision. Thus, whatever angle of deviation is found, the area of the retina which is suppressed to avoid this diplopia can be found on the troposcope with the arm set at approximately zero. In cases of variable esotropia, especially with a marked increase at near, suppression was found to continue to occur off the troposcope, although there was no evidence of suppression with or without glasses on simultaneous perception or fusion at the angle of deviation or at zero. With glasses and an additional —4.00 D. sph. for each eye, suppression was found at zero, often accompanied by a large increase in the esotropic angle, even when the glasses

gave full cycloplegic correction. This indicates an esotropia with a hyperaccommodative element, and therefore this suppression, too, should not be overlooked.

Clip-on prisms may be prescribed for two purposes: first, for relief of asthenopia, and second, to enable fusion to develop. In asthenopia, fusion is present and both orthoptics and surgical treatment for the heterophoria are contraindicated, hence the orthoptist rarely sees these cases. It is the second purpose which concerns the orthoptist mainly. Prisms have been described as a "mere crutch," but in learning to walk a crutch is invaluable. Thus it is in the development or re-education of fusion. In cases of small amounts of deviation with a negligible accommodative element in which binocular single vision cannot be obtained with the fusion amplitudes developed on the troposcope, the wearing of a prismatic correction to obtain approximate orthophoria can allow fusion to develop with full everyday usage. This is especially true if there is an associated hypertropia. When well established, the good fusion can overcome a gradual or complete decrease in the prismatic correction, as in the following case:

Bonnie first attended the clinic when she was 4 years old. She had a history of alternating esotropia, with onset when she was 3. Her visual acuity was 20/20, each eye, with glasses. Results of prism cover test showed 35△ esotropia, 6△ right hypertropia for distance, 35△ esotropia at near with glasses; 40△ esotropia, 6△ right hypertropia for distance, 45△ esotropia for near without glasses. The Worth dot test elicited alternate suppression. On the troposcope, her subjective and objective angles were both +45△, and she had 4△ right hypertropia. She had too much suppression for fusion.

Treatment consisted of two surgical operations, each accompanied by preoperative and postoperative orthoptic treatment. At the age of 6 years, results of the prism cover test were 8△ esotropia for near and 4-6△ esotropia for distance with glasses. The Worth dot test revealed homonymous diplopia. The troposcope readings were: angle, +5△; fusion, +5△, with 20△ of convergence and 6△ of divergence. In view of the lack of binocular vision in spite of the small deviation, prisms of 3 base out for each eye were prescribed. These were worn for four months, with monthly orthoptic checks. On each visit full binocular single vision was present with prisms, but it was very unstable and diplopia occurred without the prisms. After four months the prisms were accidently broken. On examination at that time, esophoria was well maintained, and so the prisms were discontinued. Five months later results of the prism cover test showed 4△ esophoria for distance, 6△ esophoria at near with glasses; 10△ esophoria for distance, 15△ esophoria at near without glasses. The Worth dot test elicited fusion with and without glasses and +15△ without glasses; fusion was elicited at zero, with 40△ of convergence and 10△ of divergence. This functional cure probably would not have been obtained without the temporary prisms.

It is not always possible for binocular single vision to be maintained without prisms, but the wearing of them may easily make possible a functional result eventually and change the original prognosis.

Jeannie first attended the clinic at 11 years of age. The date of onset of her squint was unknown; her parents had not noticed anything amiss. Her visual acuity was 20/20, each eye, with no refractive error. Results of prism cover test showed 15△ esotropia and 4△ right hypertropia for distance, and normal ocular movements at near; alternating esotropia with preference to fix with the right eye was evidenced by cover test. Results of the Worth dot test showed suppression in left eye or alternation. On the troposcope, her objective angle was +18△, 2△ right hypertropia; her subjective angle was +14△, and she had no fusion. After six orthoptic treatments good simultaneous perception was developed and fusion on the troposcope obtained. Results of the Worth dot test showed suppression in the left eye or homonymous diplopia. Diplopia with a spot light could be overcome with prisms, so prisms of 4△ base out, each eye, and 2△ base down, right eye were worn for one month. At that time there was still some suppression, but binocular vision was intermittent with glasses. Three more treatments were given to help overcome suppression, and prisms were continued for two more months, when results of the Worth dot test showed fusion with glasses and homonymous diplopia

without glasses. Results of the prism cover test were 6△ esophoria, no hyperphoria for distance and at near with glasses; 14△ esotropia, 2△ right hypertropia without glasses. The prisms were reduced and binocular single vision was well maintained. Three months later the patient complained of diplopia without glasses. She was expected to return for a further check within a short time.

In this case the original prognosis for binocularity was poor, considering the patient's age, uncertain history, and absence of fusion, together with alternation. However, with binocular vision now assured, only time will tell whether surgical intervention will be necessary; in view of the amount of deviation, it may well be.

Apart from their routine uses in diagnosis, such as in the prism cover test, estimation of fusion reserves, and checks for malingering, prisms can be an invaluable asset to the orthoptist. She can use them in demonstrating to parents how their child should be appreciating diplopia, in aiding fusion with home exercises such as use of the polaroid television trainer, and in many other procedures, depending on her initiative and ingenuity.

TRENDS IN REFRACTION

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CHANGES and trends, and the causes thereof, in teaching and practicing refraction may be of interest to this group of orthoptic technicians. I am inclined to feel optimistic about this phase of our work. I shall discuss my reasons under several headings.

1. Instruction in Refraction is Available to the Average Fellow

Previously, in many areas there was no one available for the teaching of refraction to the house officer except the senior house doctor who preceded him or, more often, some interested member of the staff. Even in the larger centers where teachers were available it was often the rule for one house officer to teach his junior. This sort of training can be either very good or very poor, depending upon the senior house officer, his interest in the subject, and the amount of time available to him. At best it was haphazard and followed no formal regular pattern. With the impact of the postgraduate courses throughout this country a definite routine has had to be followed in the teaching of refraction. Whether the course be of high caliber or not does not obviate the fact that the organization of the material presented is, in itself, a great stride forward. I happen to be very familiar with two such courses: The Ophthalmological Study Council, or Lancaster, course and the postgraduate course at Harvard Medical School. In both of these courses the student is also prepared by associated studies in physiological optics and laboratory work, and thus, in the overall picture there is integration. Most important of all, however, is the fact that these postgraduate courses make available to any number of candidates the opportunity for formal instruction in refraction, whereas formerly it was the privilege only of those who had already received a first-class residency.

2. Teaching of Refraction Is Becoming More Objective and More Liberal

I suspect that this trend is of great significance, and it does represent a departure from teaching as carried on previously. There has been a tendency in this country for centers to develop in their teaching their own techniques and views in refraction to the near exclusion of everything else. Now there is a strong tendency to teach all the various methods and to stress them equally. It is in the teaching of these various methods that physiological optics comes to the rescue, because it enables the student to evaluate what his teacher is telling him. In turn, he may make up his own mind in many instances, whereas formerly the material was digested for him. In acquainting the student with the testing for astigmatism, cross-cylinder techniques are now taught together with dial techniques. When the student completes the course he knows that both the dials and the cross-cylinder have much to offer but neither is that last word and that we still have to find a really good test for astigmatism. Less of refraction

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is now taught by rote. Formerly, great differences of opinion were expressed in the teaching about cycloplegia and its place in refraction. We even had the cycloplegia centers (Philadelphia) and the noncycloplegia centers (Boston). Now, I believe, both groups, those who lean toward cycloplegia and those who lean away from cycloplegia, realize that each method has its limitations, each has its advantages, and both intelligently used provide the proper way to get along with the problem. I believe, although this may sound rather childish, that the younger men no longer use drops just so their patients will know that they went to a doctor. This liberal attitude in the teaching of refraction is made possible by exposing the students to the influences of ophthalmologists from various sections of the country.

3. *Ophthalmologists Are Now Not So Reluctant to Buy New Type Refraction Equipment*

It used to be thought that a trial case was enough. The place of the refractor and the phoropter, the various electric retinoscopes, and other new instruments is now emphasized. More and more of this equipment which tends to make our work more efficient is entering the offices of the ophthalmologists. The younger men, particularly, are equipping themselves with these instruments with their wonderful arrangements of rotary prisms, cross-cylinders, Maddox rod, etc., making it possible to do a much greater number of tests in the allotted time.

4. *There Is a Difference in the Approach to Treatment*

There has been a great change in our attitude as to what to do with our refraction findings after we get through. There is less and less of stating "you have to wear these glasses because they are right for you" and more of a tendency to prescribe something that a person can wear. The old misconception that "if you don't wear your glasses something awful will happen to you" is passing out with potassium iodine. Glasses are prescribed for a purpose, either to correct symptoms or to influence a binocular imbalance. The strength of lens is determined by the purposes to be accomplished and not by the actual measure of the error.

5. *Refraction Is Being Appreciated Now By the Younger Men*

I believe that this is a very significant sign. Refraction is no longer the necessary evil in the practice of ophthalmology. Several factors are responsible for this welcome change in attitude. More well-trained ophthalmologists are settling away from the large cities. They now live next door to the people for whom they fit glasses. They trade in the stores of the people who wear their glasses. They know at the grass-root level "whether their glasses are being tolerated." They find that when patients are pleased with the glasses which their ophthalmologist has prescribed, they will come to him when they need surgical treatment for their eyes.

Further optimism is warranted, I believe, by the fact that our courses in refraction now attract not only those who are preparing for their residencies or those who are in the residencies or those who are preparing for their Board examinations but also men who have been in practice for some time and who just want to be brought up to date. When there is such optimism and demand, teaching must continually better itself.

REFRACTION AND SQUINT

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THIS lecture is on the relationship between refraction and extraocular muscle imbalance. As you all know, by the term refraction we mean the determination of the power of lenses that, when placed in front of the eye, will enable light from a distant object to be focused on the retina, with the accommodation at rest. Accommodation is the mechanism by which the eye can focus, that is, can form a clear image on the retina, of objects situated at different distances from the eye. This is brought about by contraction of the ciliary muscle, which results in an increase in the curvature of the crystalline lens.

The near reflex is a triad consisting of convergence and accommodation and miosis. Miosis does not concern us for the present. We are particularly interested in the relationship between convergence and accommodation. This relationship is never an absolute one-to-one relationship, but there are certain limits within which it can act. If the accommodation is measured in diopters and the convergence in meter angles, the accommodation-convergence relationship can be expressed as a ratio, A:C. As you remember, a diopter is the refractive power of a lens which focuses parallel rays of light at a distance of one meter, and the refractive power in diopters is the reciprocal of the focal length expressed in meters. A meter angle is the angle through which each eye has rotated from the primary position in order to fix an object at one meter. Therefore, the number of meter angles involved in convergence is the reciprocal of the distance in meters to the point of fixation. Consequently, if the accommodation is measured in diopters and the convergence in meter angles, the distance involved is the same and the reciprocals of the distance will be the same, making the relationship between accommodation and convergence comparable.

As an example, if you have an individual with emmetropia who is fixing at one meter, the convergence required is one meter angle and the accommodation required is one diopter; therefore A:C::1:1. If you have the same person fixing at a third of a meter, the convergence required is three meter angles, the accommodation is three diopters, and A:C::1:1. Therefore, a person with emmetropia and adequate accommodation maintains a constant accommodation-convergence relationship.

On the other hand, a patient with hyperopia of 3 diopters who is fixing at 1 meter requires the same amount of convergence, namely 1 meter angle, but his eyes must accommodate 4 diopters (1 diopter for the distance of 1 meter, and 3 diopters to overcome his hyperopia); therefore A:C::4:1. The same person when fixing at ⅓ of a meter requires a convergence of 3 meter angles, but his eyes must accommodate six diopters (3 diopters for the distance and 3 diopters to overcome his hyperopia); A:C::6:3 or 2:1. This is an example of how the accommodation-convergence relationship will vary in cases of ametropia when fixation is at different distances.

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Likewise, if we have a patient with myopia of 2 diopters fixing at 1 meter, the convergence is again 1 meter angle, but no accommodation is required, and A:C::0:1. However, if the same patient is fixing at ½ of a meter, the convergence required is again 3 meter angles, but the accommodation required is only one diopter (3 diopters required for the distance of ½ meter, minus 2 diopters of his myopia), and A:C::1:3. Again we have the accommodation-convergence relationship varying for different distances in ametropia.

EXAMINATION OF THE PATIENT

We have presented this background to show how refraction is related to convergence. What really interests us, however, is the relationship between refraction and muscle imbalance, so we will go on to another subject, namely the examination of a patient who has a squint, to see whether the relationship between accommodation and convergence plays any part in this squint.

As in all squint cases, the first part of the examination will involve the case history. We will not delve into this, as you all know the importance of finding out how long the squint has been present, which eye fixes, whether there is any familial history of squint, and whether it is constant or intermittent.

The second item which interests us, in the examination of a patient of this type, is the visual acuity. Each eye should be tested separately. If the patient has glasses, his visual acuity should be tested and recorded both with his glasses on and without them. You are familiar with the Snellen test chart and the "E" chart, and in cases of smaller youngsters, there is a picture chart that can be used.

The third point which interests us is the examination for ocular muscle balance. We will not discuss this subject very much either, as you are all well versed in this field. The usual tests for muscle balance should be used, such as Hirschberg's test and the cover test, and perhaps some subjective tests such as the Maddox rod test or the red-green glass test, if the patient is old enough to give a reasonable answer.

Now we turn to the item which is our main concern today: refraction. As you know, sometimes the examination of a patient wearing glasses is done without the use of drops. This matter of manifest refraction, which is the determination of the correcting lenses in ametropia without the use of cycloplegia, is not reliable in cases in which the accommodation is not well controlled by the examiner, such as the cases of children, or of persons with an anomaly of the convergence-accommodation relationship, or of individuals with excessive accommodation. For these reasons noncycloplegic refraction, although very useful in simple refractive problems, is not adequate for cases of squint, and in these cases we should do a cycloplegic refraction.

First, let us discuss what is meant by cycloplegia. Cycloplegia, by definition, is a paralysis of accommodation, that is, of the ciliary muscle. In refraction it is a temporary paralysis of accommodation, achieved by the use of certain drugs that are called cycloplegics. The most commonly used cycloplegics are atropine sulfate, which is used usually in a 0.25, 0.5, 1.0, or 2.0 per cent solution or ointment, scopolamine hydrobromide in a 0.25 or 0.5 per cent solution, homatropine hydrobromide in a 2 or 4 per cent solution, and just lately, a new drug called Cyclogyl®, in a 0.5 or 1.0 per cent solution. A side effect of cycloplegic drugs is the dilatation of the pupil, but this is not the main reason for the usefulness of cycloplegics; as we mentioned before, cycloplegia is used to paralyze the accommodation temporarily.

However, there is another group of drugs that do not paralyze accommo-

dation but do dilate the pupil. This dilatation of the pupil is called mydriasis, and the drugs used to dilate the pupil are called mydriatics. An example is Neosynephrine®, which may be used in a 1, 2 or 10 per cent solution or emulsion; Paredrine® is another example.

While we are on the subject of drugs that affect the pupil's size and accommodation, we might mention miotics. Miosis is a constriction of the pupil, and drugs that constrict the pupil are called miotics. Miotics also have a secondary action on the accommodation, that is, they often facilitate accommodation. Some examples of miotics are eserine sulfate, which is used in a 0.25, 0.5 or 1.0 per cent solution, pilocarpine nitrate or pilocarpine hydrochloride which is used in a 1, 2 or 4 per cent solution, neostigmine sulfate in a 5 per cent solution, and lately another drug, DFP, which is much stronger than the above and which is used in a 0.10 per cent or less concentrated solution. We will come back to miotics later.

To return to our cycloplegic refraction, once a cycloplegic drug has been instilled and sufficient time has elapsed for it to act, the refraction under cycloplegia is carried out. The main test to be used now is retinoscopy.

Retinoscopy is an objective method of measuring the patient's refraction by determining the focus of the rays of light which are reflected back from the patient's retina when a beam of light is thrown into the eye. This is accomplished by means of a special instrument which is called, appropriately, the retinoscope. When we say that retinoscopy is an objective test, we mean that it does not require much cooperation nor interpretation on the part of the patient. No questions are asked, and the only thing required of the patient is that he hold his eyes fairly still. Therefore, this method of examination can be carried out in very small youngsters, even some that are only months old. As you well realize, this would be of the utmost importance in examining children, who comprise the majority of our patients with accommodative squint. If the child is old enough, in addition to retinoscopy, some subjective tests may be carried out and the visual acuity may then be measured with corrective lenses.

While the child is under cycloplegia, it is sometimes helpful to measure the angle of squint again. If the squint is completely eliminated by cycloplegia, this confirms our suspicion that accommodation has played a very important part in the squint. However, one must remember that in many cases in which cycloplegia is incomplete, the squint may persist or be augmented. This is due to the fact that increased stimulation to the accommodation is necessary in order to make the eyes focus. This is accompanied by an overstimulation to convergence, and therefore the squint may be more noticeable than before the cycloplegic was instilled. If this point is not kept clearly in mind, one can sometimes be led astray by finding that the squint is more marked after the cycloplegic is instilled than before.

I might digress here for a moment to talk about the effects of miotics on squint. The use of miotics in treatment of squint is a fairly new approach to the problem. Although it was originally postulated at the turn of the century, it has not come into common usage until fairly recently. The theory regarding the use of miotics for the reduction of convergent squint is that miotics facilitate accommodation; that is, the accommodative mechanism becomes more responsive to stimulation. If less stimulation to the accommodation is required, there will be less reflex stimulation to the convergence and this will result in a diminution of the convergent squint. Some good results have

been reported by the use of this line of treatment in cases of convergent squint. The usual miotic employed for this purpose is DFP, because it requires less frequent instillations. There are some very undesirable side effects resulting from the use of miotics, especially in younger individuals. The most important of these side effects is the formation of iris cysts, which occurred in two thirds of the cases in one series. The cysts may appear very soon after the treatment has been started or later on. They have been reported as occurring as early as one week or as late as forty weeks after treatment with miotics was begun. For this reason, miotics are relatively dangerous drugs to use and one hesitates to use them on any eye that is normal in every respect except for the muscle balance.

TYPES OF ACCOMMODATIVE SQUINT

The next subject that we wish to discuss is the classification of accommodative squint. We will first talk about convergent squint, as this is the most common type and the one that is most often associated with accommodation. The usual type of accommodative squint, therefore, is an esotropia associated with hyperopia. In order to see distinctly, the child has to overcome his hyperopia, and the stimulation for accommodation in excess of that required to compensate for the hyperopia is reflexly transmitted to the convergence, resulting in an overconvergence. You are all familiar with this type of case and we will not discuss it further.

Another less common type of convergent squint associated with accommodation is the esotropia of congenital myopia. In this case, the individual's vision is blurred for distance because of his myopia. For near vision, he has to hold objects very close in order to see them distinctly. This results in an overstimulation to the convergence, without a corresponding stimulation to the accommodation for near. As the convergence doesn't make any difference in the distance vision, which is blurred already, the convergence necessary for near is retained for distance, and the squint becomes constant or noticeable, both for distance and near.

Other less common types of accommodative squint occur in certain cases of divergent squint. First, we might consider the exotropia of high acquired myopia. In this case, accommodation is not required for distance or for near; therefore, there is no reflex stimulation to convergence, and the convergence relaxes, resulting in an exotropia.

A second type of accommodative exotropia occurs in high and equal bilateral refractive errors, such as high astigmatism or very high hyperopia, which are too great to be corrected by accommodation. The individual with this type of refractive error develops a divergent squint because no improvement in the vision results from accommodation. Therefore, there is no incentive to accommodation and no reflex stimulation to convergence; hence, the eyes tend to diverge.

The third type of divergent squint associated with accommodation, or lack of it, is termed consecutive divergence. In a case of this type, the patient originally has a monocular esotropia with marked amblyopia ex anopsia. As the child grows, there is no reinforcement of convergence from binocular vision; therefore, there is a gradual abandonment of the convergent position of the eyes, leading toward a parallel position and eventually toward a divergent position. The punctum convergens basalis (PcB) becomes remote, and exotropia will manifest itself first for near. Later it spreads backward to the far point, the end result being divergence for all distances.

A fourth type of exotropia in accommodative squint occurs in mentally defective persons. These individuals, because of their low mentality, do not do much near work, and there is disuse of their convergence mechanism, resulting in an exotropia independent of the refractive error.

DISPOSITION OF THE CASE

Our last topic in this discussion is the disposition of the case. In other words, what are we going to do with this youngster who has an accommodative squint. The first approach to treatment available to us is the prescription of glasses. To the patient with hyperopia and convergent squint, we should give the full cycloplegic correction in order to eliminate the need for excessive accommodation and, therefore, the reflex excessive convergence. We may give him a cycloplegic drug to use at home for a few days, or weeks, while he is getting used to the high plus correction. As the child gets older, it is advisable to reduce the strength of the hyperopic correction to the least possible correction that will keep his eyes straightened and still maintain good vision.

To the patient with myopia associated with exotropia, we should also give the full myopic correction, so that he has to accommodate for near and therefore stimulate convergence, which tends to counteract the exotropia. It has been recommended recently that we might give such individuals more minus correction than they require, in order to increase the stimulation of convergence. This is a debatable point, and I do not believe that it has many advocates.

To the patient with high astigmatic errors in the presence of either a convergent or a divergent squint, we should give the full astigmatic correction in order to clarify the image and thus encourage fusion.

I want to mention the use of bifocals in cases in which the full hyperopic correction restores fusion for distance but the esotropia remains for near. In these cases, if we give a bifocal addition which will focus the image for near, the child will require no accommodation for near and the reflex stimulation to convergence will be minimized. A +3.00 add should be given which will focus for a distance of ⅓ meter, instead of a +2.00 which would focus for ½ meter, because children, with their short arms, usually hold things at a distance from their eyes of about ⅓ of a meter or less. We should also remember to give a fairly large bifocal segment and have it adjusted high, so that the child will not be tempted to look over the segment and thus cancel the effect of the bifocal. It is also very important to remember that after the child learns to fuse at near, the bifocal correction should be reduced and eventually eliminated completely to encourage development of a normal accommodation-convergence relationship.

Another very important factor to remember in prescribing glasses for patients with squint is that the glasses should be adjusted properly, not only for comfort but also to avoid a prismatic effect from decentration.

The other lines of treatment in cases of strabismus are orthoptics and surgical correction, as you all know. I mention them here only for completeness as they are beyond the scope of this paper.

THE MANAGEMENT OF ANISOMETROPIA

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ANISOMETROPIA of significant degree is found in about 10 per cent of cases in which refraction is done. These cases include various inequalities of hyperopia, myopia and astigmatism in all possible combinations, including antimetropia in which one eye is hyperopic and the other myopic. Anisometropia may be congenital or result from developmental changes in youth or age or be acquired from conditions which change the refraction of the cornea or lens, the length of the globe, or the position of the macula.

Anisometropia may be accompanied by binocular vision, simultaneous vision without fusion, monocular vision, or alternating vision in which one eye is used for distance and the other for near. Muscular imbalance is frequently associated with anisometropia. Duane[1] found a significant lateral deviation in half of his cases of anisometropia, and in about 25 per cent of the total he found manifest squint, convergent in 11 per cent and divergent in 14 per cent. He noted a hyperphoria of 1△ or more in one third of these patients. In a personal study of 200 consecutive cases of anisometropia in which the difference in refractive power in the vertical meridian was 0.75 D. or more, I found that a hyperphoria of 0.5△ or more for either distance or near was present in 80 per cent, and of 1△ or more, in 50 per cent.[5]

ACCOMMODATIVE AMBLYOPIA

In persons with anisometropia, one eye may be amblyopic to some degree. Since the power of accommodation is normally equal for the two eyes, such a person can accommodate for a clear image in only one eye. The other eye receives a blurred image both for distance and for near, and accommodative amblyopia tends to result. Hence, equal corrected acuity is significantly commoner in cases of anisomyopia than in anisohyperopia, and the disparity in the corrected acuity of the two eyes reaches higher values in the latter condition, especially when it is associated with astigmatism.[3] In the higher degrees of anisohyperopia the child accommodates for the less hyperopic eye, but in lower degrees thereof, the more hyperopic eye may be favored because of ciliary tone, which in a child of 7 years may reach 1.50 D. Even 0.50 D. of anisohyperopia may produce some amblyopia, which is usually overcome simply by wearing the proper optical correction.

Case 1. A girl, aged 10 years, was referred to us because of poor vision in the left eye. Her unaided acuity was R.E., 20/13; L.E., 20/40. Under atropine cycloplegia the correction was R.E., +0.50 sph., 20/13; L.E., plano, 20/20. To allow for ciliary tone a symmetrical reduction of 0.50 D. was made so that the prescription ordered was R.E. plano, L.E., −0.50 sph.

Children adapt themselves readily to any type of anisometropic correction. If one eye is amblyopic, its vision is generally improved by temporary occlusion of the better eye, as in the following examples:

Case 2. A girl, aged 9 years, was discovered in the school screening test to have poor vision in the left eye. Her unaided acuity was R.E., 20/15; L.E., 20/100. Under atropine cyclo-

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plegia the correction was R.E., +1.25 sph., 20/15; L.E., +3.50 sph., 20/50. The prescription was reduced symmetrically by 1.00 D. and a Bel-occluder was ordered for the right lens. After 4 months the corrected vision of the left eye was 20/25.

Case 3. A boy, aged 13 years, was brought to the office because of a minor injury to the right upper lid. Results of examination disclosed that his unaided acuity was R.E., 20/40, L.E., 20/200. Under atropine cycloplegia the correction was R.E., −0.75 sph., 20/13; L.E., +2.00 sph., +1.50 cyl., X 105, 20/50. Without glasses and with the right eye fixing, there was left hyperphoria 1.5△ for distance, none for near. In the prescription, −0.25 sph. was added symmetrically for ciliary tone and the prism was incorporated in the left lens. The prism corrected the hyperphoria for distance and at the same time neutralized the base-up prism created by the lenses on looking down. Three months later the corrected vision was R.E., 20/15; L.E., 20/30 minus. After a month's use of a Bel-occluder over the right lens, the corrected acuity of the left eye was 20/20. By the following year its corrected vision had improved to 20/15 minus a few middle letters.

RELATION OF INTRINSIC HYPERPHORIA TO ANISOMETROPIA

The principal cause of discomfort in anisometropia is the artificial heterophoria created whenever the eyes move from the primary position, especially the induced hyperphoria when looking down. The other disturbing factors are of minor import. In axial ametropia the aniseikonia is diminished by the indicated correction. The ocular accommodation is affected unequally by the anisometropic lenses, but a disparity of 10 D. produces an accommodative difference of only 0.75 D. The prismatic disparity and the size difference can be minimized by proper coflexures. The stronger plus or the weaker minus lens should have a relatively flat back surface, whereas the back surface of the weaker plus or stronger minus lens should be deeply curved. An additive effect is secured by making the stronger minus lens of safety thickness, which also reduces the fragility of the lens. The anisophoria induced by 1 D. of anisometropia can be thus compensated, which may reduce the prismatic effects to the limit of tolerance. In anisomyopia of high degree, in both children and adults, the induced anisophoria can be neutralized by a suitable slab-off in the more minus lens. This method is undesirable in anisohyperopia as the slab-off would need to be placed on the less convex lens with consequent annoyance to the better-seeing dominant eye. A fused compensatory base-down prism in the more convex lens is unobjectionable but, though available some years ago, is unfortunately no longer manufactured. Contact glasses are an excellent, and sometimes the only, solution for anisometropia.

No form of optical compensation for anisometropia should be prescribed without first considering the influence of any intrinsic hyperphoria that may be present for distance or for near. Hyperphoria is usually incomitant, that is, greater in the primary position than at the reading level of the eyes, or vice versa. White[8] demonstrated that the involved muscle commonly shows a paretic attribute in even the lesser degrees of hyperphoria. Friedenwald called this condition anisophoria and held that as little as 0.5△ of vertical anisophoria might be clinically significant. My own studies showed that in two out of three cases of anisometropia the intrinsic anisophoria simplified the handling of the case and made the need for optical compensation unnecessary. If the induced anisophoria created by the lenses corrects the intrinsic anisophoria of the eyes, a slab-off would then be disturbing rather than helpful. When hyperphoria is present in the primary position but not at the reading level, the prism that corrects the distant hyperphoria neutralizes or mitigates the prismatic differential of the lenses.

DETERMINATION OF INTRINSIC HYPERPHORIA

The tests for hyperphoria must be done in the following manner to reflect accurately the situation present:

1. The dominant eye is determined while the patient is wearing his corrective lenses.
2. The hyperphoria tests are made with the corrective lenses removed.
3. A dark room facilitates the examination, and a large spotlight, 2.5 cm. in diameter, is advisable for distance fixation.
4. The red glass is placed over the dominant eye; a white ribbed Maddox rod over the other. The measuring lenses are placed over the Maddox rod.
5. Hyperphoria for distance is determined with the eyes in the primary position and for near with the patient holding a small fixation light in the habitual reading position with the eyes turned down about 20 degrees.

The simplest and least equivocal of the many methods of determining ocular dominance is the sighting test. A hole, the size of a quarter, is placed in the center of a sheet of cardboard or darkened x-ray film, the size of standard typewriter paper. The patient, holding the sheet at arm's length with both hands, gazes through the hole at the distant fixation light. While maintaining this gaze, the sheet is gradually brought against the face. The hole will then overlie the dominant eye.

Hyperphoria is not affected by accommodation, and errors in its determination are avoided if corrective lenses are not used. Lenses complicate the picture because of the differential prismatic effect of an anisometropic correction. Moreover, the trial lenses themselves can introduce adventitious prismatic effects. Schilling found decentration errors of 0.5△ to 3△ in 43 per cent of nonprecision lenses and in 13 per cent of so-called precision lenses.

The natural fixation can be altered by the Maddox rod or the applied prism. In the absence of a test prism one fixes usually with the eye looking through the red glass rather than that behind the Maddox rod, but the obscuration produced by the addition of a prism can shift the fixation to the opposite eye. To assure continued fixation of the eye behind the red glass, the measuring prisms must be placed over the Maddox rod.

In a personal analysis of 100 cases of hyperphoria, measurements were made for distance and for near with each eye fixing in turn.[6] The change of fixation caused unequal measurements in 55 per cent, with the disparity being equal to, or exceeding, 1.5△ in 20 per cent of the involved group. The variation simulates the primary and secondary deviations characteristic of paresis. The eye that when fixing elicits the greater amount of vertical imbalance has the involved muscle; this may be either the dominant or the nondominant eye. The following are characteristic cases:

Case 4. A woman, 56 years old, was given a refraction. Findings were R.E., +2.00 sph., +1.50 cyl., axis 30, 20/20; L.E., −1.25 sph., +4.50 cyl., axis 135, 20/25. The right eye was dominant. The presbyopic addition was 2.50 D. The difference in the vertical power of the lenses was 1.83 D., which would induce a prismatic effect of 1.5△ base up, right eye, at the reading level. With the right eye fixing, no hyperphoria was present for distance and 2△ left hyperphoria was present for near. With the left eye fixing, the measurements were 1△ left hyperphoria for distance and 4△ left hyperphoria for near. The findings with the dominant right eye fixing indicated that the anisometropic lenses corrected the intrinsic hyperphoria for near within 0.5△, the physiological tolerance, so that no optical compensation was necessary. The measurements with the left eye fixing demonstrated that the left eye had the involved muscle and that it was a depressor. With fixation of gaze at the near light, the hyperphoria for near tended to increase as the patient turned her head to the left, thus causing adduction of the left eye, which showed that the left superior oblique muscle was involved.

Case 5. A woman, aged 55 years, was given a refraction, with the following results: R.E., +4.75 sph., +1.75 cyl., axis 15, 20/15; L.E., +2.25 sph., +1.50 cyl., axis 165, 20/15. The left eye was dominant. The presbyopic addition was 2.25 D. With the dominant left eye fixing, a right hyperphoria of 2△ was present for distance and no hyperphoria for near. With the right eye fixing, no hyperphoria was found for distance or for near. In distance fixation on turning the head to the right, which caused abduction of the left eye, the right hyperphoria tended to increase, demonstrating that the left superior rectus muscle was affected. The difference in vertical power of the lenses was 2.75 D., which would cause a prismatic difference of 2.2△ base up, right eye, at the reading level. By adding to the right lens the 2△ base down indicated when the dominant left eye was fixing for distance, the anisophoria induced by the lenses was neutralized and no other optical compensation was needed.

OPTICAL COMPENSATION FOR ANISOMETROPIA

The data obtained with the dominant eye maintaining fixation are of primary importance. Results of a comparison of these data with the data obtained following a change of fixation will indicate the involved muscle and so will supply information that may be of clinical interest. Occasionally the intrinsic vertical muscle balance is normal and the prismatic differential at the reading level must be optically compensated. The methods employed are illustrated in the following cases:

Case 6. A salesman, aged 59 years, complained of progressive impairment of the vision in his left eye. Results of examination disclosed nuclear sclerosis of the left crystalline lens. Findings on refraction were: R.E., +2.75 sph., +1.00 cyl., axis 10, 20/15; L.E., −1.75 sph., −2.25 cyl., axis 72.5, 20/25. The right eye was dominant. The presbyopic addition was 2.25 D. With the dominant right eye fixing, no hyperphoria was present for distance or for near. The difference in vertical power of the lenses, 5.6 D., caused a prismatic differential at the reading level of 4.5△ base down, left eye. This was compensated by a slab-off of the same amount in the left lens of the Univis bifocals ordered.

Case 7. A saleswoman, aged 58 years, was given a refraction and the findings were: R.E., −6.00 sph., −2.25 cyl., axis 22.5, 20/20 plus; L.E., −9.50 sph., −1.25 cyl., axis 135, 20/20 plus. The left eye was dominant. The presbyopic addition was 2.25 D. Her distance correction had been remarkably constant during 14 years of observation. The difference in vertical power of the lenses was 2.25 D., producing a prismatic differential at the reading level of 1.8△ base down, left eye. Univis bifocals with bilateral slab-offs were ordered so as to reduce the displacement in the reading field effected by strong minus lenses. The upper limit of slab-off, 5△, was placed in the left lens, while that for the right lens was 3.2△. This difference neutralized the prismatic differential of the lenses at the reading level.

Case 8. A woman, aged 46 years, was given a refraction, with the following results: R.E., +0.50 sph., 20/13; L.E., +0.75 sph., −1.25 cyl., axis 175, 20/13. The right eye was dominant. The presbyopic addition was 1.5 D. With the dominant right eye fixing, no hyperphoria was present for distance or for near. The difference in vertical power of the lenses was 1.0 D., causing a prismatic differential of 0.8△ base down, left eye, at the reading level, just enough to cause discomfort with bifocals unless compensated. Since the lower limit of slab-off is 1.5△, this was achieved with dissimilar segments: R.E., Orthogon D., L.E., Univis D. With 1.5 D. in the reading addition, the 5 mm. difference between the optical radiuses of the segments caused 0.75△ base down, right eye, which effectively neutralized the prismatic differential caused by the distance correction. The refraction of the left eye, originally identical with that of the right eye, changed after a traumatic erosion of the left cornea.

Dissimilar segments provide the simplest and least expensive method of optical compensation in presbyopic cases. As an alternative, the more expensive and less readily procurable prism segment may be used.

When the prism correcting the hyperphoria for distance is insufficient to annul the prismatic differential at the reading level, dissimilar segments may be used for complemental compensation as in the following cases:

Case 9. A seamstress, aged 53 years, was given a refraction, the results of which were: R.E., +2.00 sph., 20/13; L.E., −2.25 sph., +6.00 cyl., axis 50, 20/40. The right eye was

dominant. The presbyopic addition was 2.0 D. When she was tested without her glasses and with the dominant right eye fixing, a left hyperphoria of 1△ for distance was noted; there was no hyperphoria for near. The difference in the vertical power of the lenses was 2.25 D., causing a prismatic differential of 1.8△ base up, right eye, at the reading level. The inclusion of 1△ base up in the left lens left 0.8△ to be compensated. This was achieved by prescribing dissimilar segments: R.E., Orthogon D; L.E., Univis D.

High anisomyopia may be associated with a true paresis in the more myopic eye. If the separation of images in the reading field is aggravated by the lenses, a presbyopic individual can be rendered comfortable by wearing a bifocal only for the less myopic eye. The next case exemplifies this situation.

Case 10. A woman, aged 61 years, had always had to keep the left eye closed when reading. Results of refraction were: R.E., +0.75 cyl., axis 5, 20/15; L.E., −10.00 sph., −1.75 cyl., axis 180, 20/50 plus. Examination disclosed a paralysis of the left superior rectus with a contracture of the left inferior rectus, causing a left hypophoria of 15△ in both the primary position and the downward gaze. A bifocal with the presbyopic addition of 2.5 D. was ordered for the right eye, but only the distance lens (in coated thinlite for better appearance) was ordered for the left eye. The absence of the presbyopic addition fogged the left eye for near so that she was enabled to read without closing it.

The vertical muscle balance at the reading level is of more importance than that for distance as the customary gaze is below eye level. The prescription should be influenced by this consideration, as exemplified in the following case:

Case 11. A woman, aged 47 years, was given a refraction, with the following results: R.E., +3.50 sph., +0.50 cyl., axis 160, 20/13; L.E., +5.75 sph., +0.75 cyl., axis 165, 20/15. The right eye was dominant. The presbyopic addition was 1.5 D. With the dominant right eye fixing, the intrinsic anisophoria measured for distance, 1.5△ base down, left eye; and for near, 2.75△ base up, left eye. As the lenses induced 2△ base up, left eye, at the reading level, the rest of the intrinsic anisophoria for near was corrected by dissimilar segments: R.E., Ultex E.; L.E., Ultex B.

ALTERNATING VISION IN MYOPIA

In some cases of high anisomyopia in which the corrected acuity of each eye is good, it is wise to abandon the attempt to secure binocular vision with its attendant difficulties and to promote alternating vision instead. By this means one eye is adapted to distance vision and the other to near vision.[4] With the use of one eye at a time, the demand for balanced muscular activity is annulled and symptoms arising from incoordination cannot develop. Because of the unequal progression of myopia, many patients with this disability have developed a natural tendency to alternate vision. Whether or not this tendency exists can be determined by the Worth four-dot test. Let us assume that the patient has the habit of using the right eye for distance and the left eye for near. With the red-green spectacles in place (the red glass over the right eye and the green glass over the left eye), the patient will call the white dot red at distance and green at near. The fixing eye for distance can be ascertained by the sighting test; that for near, in testing the near point of convergence. Every effort should be made to maintain binocular vision in children and young adult patients, as an undercorrected eye may then turn out. Likewise, when binocular vision is well established, the substitution of alternating vision should not be attempted; if tried, it will not be tolerated. When clearly indicated, the arrangement for alternating vision improves the appearance of the glasses and also avoids the need for bifocals. The following examples illustrate its use:

Case 12. When first examined, at the age of 31 years, a woman had the following results from refraction: R.E., −0.50 sph., −3.00 cyl., axis 42½, 20/15; L.E., −8.00 sph., −.75 cyl., axis 152½, 20/15. In the prescription ordered, the left sphere was lessened 2.50 D. These

glasses gave the patient complete comfort, and she wore them until, at the age of 44, she noted that her vision was not as clear as formerly. Re-examination showed a change of refraction to R.E., −0.50 sph., −2.25 cyl., axis 40, 20/15; L.E., −9.50 sph., 0.75 cyl., axis 135, 20/15. The left lens was given the same addition as before, the prescription being −7.00 sph., −0.75 cyl., axis 135, with which she read 3-point type at 14 inches. Three years have elapsed, and she still finds the glasses highly satisfactory.

Case 13. A woman singer, aged 36 years, found her glasses annoying and unsightly. They were correct: R.E., −4.75 sph., +3.00 cyl., axis 92½, 20/25; L.E., −2.25 sph., +3.50 cyl., axis 67½, 20/20. A change of the right lens to −2.25 sph., +3.00 cyl., axis 92½ added to both her ocular ease and appearance.

Case 14. A grocer, aged 49 years, had three pairs of glasses, none of which he could use. Findings at refraction were R.E., −2.75 cyl., axis 37½, 20/30; L.E., −11.00 sph., −2.50 cyl., axis 157½, 20/30. The left lens was changed to −8.50 sph., 2.50 cyl., axis 157½, with which he read 4-point type at 13 inches. He could now read the scales and wait on customers efficiently.

Case 15. An optometrist, aged 51 years, consulted me because of troublesome heterophoria. Results of refraction were R.E., −6.00 sph., 0.75 cyl., axis 70, 20/13; L.E., −4.50 sph., −0.50 cyl., axis 75, 20/13. The left eye was dominant. The presbyopic addition was 2.0 D. Tested with the indicated correction for distance and near, he was found to have an exophoria of 14△ for distance and 28△ for near. Results of the vertical muscle balance test, when he was not wearing glasses and with the dominant left eye fixing, revealed 2.5△ right hyperphoria for distance and for near. The right eye was fitted for near by reducing the sphere by 2.25 D.; the distance correction of the left eye was not changed. By suppressing binocular vision his muscular difficulties were eliminated.

This method may also be applied with occasional success to cases of troublesome vertical diplopia with presbyopia.

Case 16. Following a skull fracture, a man 53 years old, had paresis of the left inferior rectus muscle. Results of refraction were R.E., −0.50 sph., −0.50 cyl., axis 105, 20/13; L.E., −0.25 sph., +0.50 cyl., axis 80, 20/13. The left lens was not changed, as the left eye was to be used for distance vision only, but the prescription for the right lens was changed to +2.00 sph., +0.50 cyl., axis 105 for near vision. With these glasses his discomfort disappeared.

When useful vision is present in only one eye, patients should be relieved of an unsightly anisometropic correction from which no benefit is derived, whether squint is or is not present. Even if the corrected vision of the amblyopic eye is 20/200, it is better to prescribe balanced lenses, as illustrated in the next case:

Case 17. A woman, aged 57 years, had recently been examined by a competent refractionist, who first had prescribed R.E., −12.00 sph., −1.00 cyl., axis 90, 20/200; L.E., −5.25 sph., −1.50 cyl., axis 75, 20/30. When she complained, he substituted a plano glass for the right lens, but that proved equally unsatisfactory. After the right lens was changed to −5.50 sph., her eyes were comfortable, and they remained so during five years of observation.

CONTACT-LENS CORRECTION

When contact lenses are used for the correction of anisometropia, it is often necessary to use spectacles, also, in order to provide the best possible visual acuity. The contact-lens correction is rarely exact, as plastic material is more difficult to work with than glass, yet the tolerance of the optic radius is only 0.001 inch. In monocular aphakia and extreme anisomyopia, contact lenses provide the only effective solution. The following case, reported by Dr. Charles Littwin,[7] is apropos:

Case 18. An engineer, aged 30 years, complained of visual difficulty with his glasses. The prescription was R.E., −13.00 sph., 20/50 minus; L.E., −0.25 cyl., axis 180, 20/20. Iseikonic lenses were next prescribed. Though these spectacles gave him fusion and comfort in the primary position, he experienced distortion and diplopia when he moved his eyes. A contact lens gave the right eye acuity of 20/50 plus, and with a +1.00 sph. in the spectacle lens, the acuity was improved to 20/30 plus. The contact lens permitted fusion in all directions and completely relieved his asthenopia.

By means of contact lenses, Gyoerffy[2] restored binocular vision to 20 of 25 subjects with monocular aphakia and normal acuity in the fellow eye. Whereas a spectacle correction causes an image enlargement of 25 per cent, the contact lens reduces the enlargement to about 8.5 per cent, which is generally tolerated. This magnification can be eliminated by the addition of +6 D. to the contact lens correction and the neutralization of this overcorrection by a spectacle lens of −6.5 D. at 12.8 mm. from the cornea. This technique is especially applicable to patients whose normal eye is moderately myopic. By this method the image in the aphakic eye is reduced by the reverse telescopic principle. A nonirritated aphakic eye has no difficulty in tolerating a contact lens. The following report illustrates such a correction.

Case 19. While playing basketball, a youth of 18 received a blow over the right spectacle lens. The shattered lens caused a jagged cut through the cornea, with a sequential prolapse of the iris and a traumatic cataract. At operation the iris prolapse was excised, the lens expressed after partial removal of its capsule, and the cornea sutured. A persistent capsular membrane was later needled and a clear pupil resulted. Prior to the accident his spectacle correction was R.E., −2.50 sph., 20/15; L.E., −3.75 sph., 20/15. After the accident the spectacle correction of the right eye was +7.25 sph., +2.75 cyl., axis 135, 20/25. The contact lens ordered was not sufficiently overcorrected, but as he was comfortable, no change was made. His refraction with the contact lens in place was R.E., −1.50 cyl., axis 120, 20/15 plus. The near addition was +2.50 D., with which he read 4-point type at 16 inches. A Fulvue bifocal was ordered for the right eye in safety-thickness tempered glass, and a laminated safety glass (Motex), −3.75 sph., was prescribed for the left eye. The orthoptic technician reported that with the correction he had grade-3 fusion, fine fusional amplitude and stereopsis.

INCREASING HYPERPHORIA

Just as uncorrected myopia or hyperopia tends to produce an adverse effect in the lateral phorias, the frequent association of vertical phoria with anisometropia is probably another, though less understood, expression of abnormal physiology. In axial myopia the larger and heavier globe demands more of its superior rectus muscle and the activity thereof may be modified further by a shift in the arc of contact and in the ocular center of rotation. In refractive myopia, the consequent aniseikonia could give an anisophoria like that of a size lens, with a hyperphoria manifest for near but not for far. The eye with the higher magnification must turn down more than its fellow to fixate a point 20 degrees down. A correcting prism alters neither the aniseikonia nor the innervation impulse to anisophoria. The following case report exemplifies the increase in hyperphoria that occasionally follows the constant wearing of a prismatic correction.

Case 20. A male office worker habitually returned every three years with the same complaint: that his eyes tired and did not focus fast enough or sharply enough. When first examined, at the age of 28 years, his refraction was R.E., −2.25 sph., −1.75 cyl., axis 170, 20/15; L.E., −0.75 sph., +2.00 cyl., axis 97.5, 20/25. At that time he had lateral orthophoria for distance, 6.5△ exophoria for near. With the right dominant eye fixing, he had 1.5△ left hyperphoria for distance, and 1△ left hyperphoria for near. The vertical imbalance steadily increased over the years, and at each examination the indicated prism was ordered. At the most recent visit, the patient, now 45 years old, had the following findings on refraction: R.E., −2.25 sph., −0.75 cyl., axis 75, 20/15; L.E., +0.75 cyl., axis 100, 20/25. With the dominant right eye fixing, there was now 7△ left hyperphoria for distance and 5△ left hyperphoria for near. A total of 7△ was prescribed, 3△ base up, right eye; 4△ base down, left eye. This compensated the 2△ base down, right eye, induced by the anisometropia at the near point and fully corrected the intrinsic vertical imbalance both for distance and for near.

CONCLUSIONS

1. Adults with anisometropia should be tested routinely for intrinsic anisophoria, as the two conditions are com-

monly associated. Only after the intrinsic anisophoria is determined can one know whether optical compensation is or is not required.

2. The intrinsic hyperphoria should be tested without corrective lenses and with the dominant eye fixing for distance and for near. Both the Maddox rod and the measuring prisms must be placed over the nondominant eye to assure continued fixation by the dominant eye.

3. In a hyperphoria for distance only, the prismatic correction should be prescribed when it is helpful in neutralizing the anisophoria induced by anisometropic lenses, as often happens, but not otherwise.

4. In cases of anisometropic myopia in which fusion is poor but there is good correctible acuity in each eye, the more myopic eye may be fitted advantageously for near vision and the less myopic eye for distance.

5. Contact glasses are an excellent, and sometimes the only, solution for anisometropia in cases of monocular aphakia and monocular high myopia. Supplementary spectacles usually add to the visual efficiency of the correction.

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NEUROANATOMY FOR THE ORTHOPTIST

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THE orthoptist is working every day with the end effects of a very complex, and only partially understood, mechanism of ocular movement. Certain facts about the nerve pathways concerned with various types of ocular rotation are well established; some hypotheses on the subject are the source of endless controversy, and many are entirely theoretical. We shall attempt to deal with known pathways insofar as possible.

The basic unit of which the nervous system is composed is called a *neuron*. This is a single specialized cell with long processes. The neuron doctrine, a basic law in neuroanatomy, states that each nerve cell, with its processes, has its origin in a separate embryonic cell and in its adult form still represents a cellular unit. Due to their specialized construction, each of the extensions, or processes, of the neuron conducts only in one direction. The incoming processes, the dendrites, are usually the shorter ones; those carrying an impulse away from the cell, the axons, are ordinarily the longer. Each neuron receives one or many impulses by way of its dendrites, and discharges them to produce certain effects on the cells which its axon contacts.

Neurons are arranged together in chains and systems of conduction much like a complex telephone exchange. When the axons of a number of nerve cells run together, as in a telephone cable, this grouping is called a nerve pathway. The pathway need not consist all the way of single axons; it can be composed of a series of nerve cells. When a bundle of axons leaves the central nervous system to run out to a muscle, the pathway then becomes a peripheral nerve.

The point of junction at which the axon of one nerve cell contacts the dendrite of another to transmit an impulse is called the *synapse*. It is this junction which determines the direction of flow stimulation – from axon to dendrite only – like a one-way valve, but the exact mechanism of conduction across the synapse is still in doubt.

In order for the multitude of impulses from the central nervous system to produce an effect on movement of a muscle, there must be a final common pathway leading to the muscle itself. This is supplied by what is known as a *motor nerve*. If the muscle is derived embryologically from somites, the nerve is known as a "somatic efferent" – efferent because it conducts away from the central nervous system. The oculomotor (III), trochlear (IV), and abducens (VI) nerves which lead to the extraocular muscles are of this type. Their cell bodies lie within the central nervous system and their axons, running by way of the respective nerves, innervate the muscles.

One might conceive of the extraocular muscle as the buck private, the motor nerve as the corporal. Each is capable of functioning only if orders come from the higher echelons, and as one ascends the scale toward higher rank – "through channels," as it were – the complexity of organization increases

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with a corresponding increase in independent activity at each level.

The cell bodies of the neurons which make up the respective oculomotor, trochlear and abducens nerves are gathered together in groups known as *nuclei*. These nuclei lie in paired lines within the brain stem: the oculomotor nuclei, farthest forward at the level of the midbrain; the trochlear, just behind on each side; and the abducens, farther down the brain stem at the level of the pons. The organization of the nerve cells within the oculomotor nuclei has been the subject of controversy, but we shall retain the neuroanatomical concept until it has been proved wrong by neuroanatomical studies.

At the higher end of the paired oculomotor nuclei lie the cells which innervate the levator of the upper lid; next are found those for the superior rectus muscle. These lead to the muscles on the same side; that is, the fibers from the left oculomotor nucleus supply the corresponding levator and superior rectus muscles of the left eye. Next in line lie the nerve cells for the medial rectus muscles. These go to both sides. The median connection between the two oculomotor columns is known as the nucleus of Perlia and is believed to be concerned with convergence. The nerve fibers to the inferior oblique muscle are both crossed and uncrossed; those to the inferior rectus are crossed to the other side. The trochlear nerve is entirely crossed from one side of the brain stem to the opposite superior oblique muscle. The fibers from the abducens nucleus all go to the lateral rectus muscle of the same side.

No one of the nuclear groups just described is capable of independent action – just as no one ocular muscle can be moved by itself. There must be a basic mechanism by which they may function together. For example, when the right lateral rectus turns the right eye to the right, the left medial rectus turns the left eye to the right also – producing right conjugate deviation, or dextroversion. Therefore, a connecting pathway between the various ocular nuclear groups is necessary to produce versions. This is supplied by paired bundles of nerve fibers which extend up and down the brain stem along with the nerve nuclei. These bundles are called the median longitudinal fasciculi. These fasciculi are very primitive, from the phylogenetic point of view, and are present in animals as far down the vertebrate scale as the shark and salamander. In addition to linking together the contracting muscles in right dextroversion, they also correlate the relaxation of the antagonistic muscles– the left lateral rectus and right medial rectus. This provides the final correlative pathway for reciprocal innervation of the ocular muscles.

All of the versions, regardless of the level of control over them, must utilize this final correlated pathway consisting of the extraocular muscle nuclei plus the multiple interconnections of the median longitudinal fasciculi.

The most primitive level of control over this final pathway for versions arises from the vestibular system. From the semicircular canals of the inner ear, impulses are carried by way of the vestibular nerve to end in certain groups of nuclei in the pons. These nuclei in turn give rise to fibers which intermingle in the median longitudinal fasciculi to reach the nuclear cells of the nerves to the extraocular muscles. Thus, when the head is rotated to the right, fluid in the right horizontal semicircular canal moves forward by inertia. This action of fluid is translated, by way of the vestibular nerve, vestibular nuclei, and median longitudinal fasciculi, into a conjugate movement to the left, or levoversion. The result of such a mechanism is to make the eyes re-

sistant to changes in movement of the head, and to maintain the individual's orientation in space. Conjugate movement of the eyes can be produced in any given direction, provided that the semicircular canals are placed in the proper position by changing the position of the head. One sees a nonphysiologic demonstration of this function when a patient is rotated, producing a repeated jerking movement of the eyes, conjugate in type. This is called vestibular nystagmus, and the slow deviation component is the result of stimulation of the semicircular canals.

Another portion of the vestibular system is known as the otolith apparatus. This part of the inner ear functions to maintain the position of the eyes with respect to changes in position of the head in relation to gravity. For example, in animals with eyes laterally placed, this is the system which acts to raise the left eye and lower the right eye when the head is tilted to the left. The impulses apparently go by way of the median longitudinal fasciculi to the vertically acting muscles only – not to the medial and lateral rectus muscles. In man the following reactions occur: upward deviation of the eyes when the head is flexed, downward turning of the eyes when the head is extended, and conjugate torsional movement, or cycloversion, when the head is inclined to either shoulder.

It should be emphasized that the ocular movements just discussed are all at a purely involuntary or reflex level of function – that is, their occurrence is independent of the will.

The next higher level of control over the eye movements in man arises in the cerebral cortex. Visual impulses are received by a specialized area of the occipital cortex known as area 17, or the striate area. These visual impulses are translated into visual fixation reflexes by connections from area 17 into the surrounding occipital motor areas 18 and 19. They are not conscious movements in the usual sense of the term, but result from the conscious fact of vision itself.

The most marked example of such movement occurs in the phenomenon known as optokinetic nystagmus. If a drum with vertical stripes is rotated to the right across the patient's field of gaze, he will follow it up to a certain point, and then his eyes will jerk back, only to follow it over and over again. This action is reflex in type and the patient is not aware that he is doing it. Vertical movement also can be produced if the drum is rotated in the appropriate direction. Pathways are known to pass from areas 18 and 19 down to the brain stem to produce the conjugate movement. They utilize the lower levels of control by discharging through the median longitudinal fasciculus system. The fibers which result in horizontal movement come down the brain stem to end in or near the abducens nucleus. The abducens nucleus, in turn, is connected through the median longitudinal fasciculus to the part of the oculomotor nerve concerned with the opposite medial rectus. Thus a conjugate deviation results.

The motor fibers from the occipital lobe which produce vertical movements course down and enter the brain stem at a higher level – that of the oculomotor nucleus. From here the interrelations of the various muscles take place through the median longitudinal fasciculi.

There are two clinical facts which support this arrangement of a difference in level for the termination of horizontal and of vertical movement impulses. The first is a condition known as internuclear ophthalmoplegia. Here the median longitudinal fasciculi are interrupted between the abducens nucleus and the oculomotor nucleus.

When the patient looks to the right, the right eye abducts but the left eye fails or is weak in adduction – the movement is no longer conjugate. The reverse occurs when the gaze is to the left. Obviously, the impulse reaches the level of the abducens nucleus but cannot ascend the median longitudinal fasciculus to the opposite medial rectus muscle. You might say, "How do you know there isn't a paralysis of the medial rectus muscles?" This is answered by testing convergence, which the patient is able to perform, showing that the medial recti are capable of functioning and also showing that impulses for convergence enter at the midbrain level.

The second clinical fact is supplied by cases in which there is damage to the surface of the midbrain – such as by a tumor of the pineal gland. Vertical eye movements are interfered with, but as long as there is no deep involvement of the pathways leading down to the level of the abducens nucleus, horizontal movement is intact.

The optokinetic nystagmus just discussed is a more marked manifestation of a psychomotor reflex that is continuous in its effects in daily life. This is called the fixation reflex. If the image of an object moves off the macula, this reflex automatically brings the foveal vision back on the object.

Thus far we have confined our discussion to the versional or conjugate movements. Less is known of the anatomical basis for disjunctive, or vergence, movements. It is likely that the same region of the occipital cortex (areas 18 and 19) furnishes the site of integration of impulses which keep the eyes adjusted at the proper angle to maintain single binocular vision. It is also likely that the integration of impulses for fusion is located here as well. The usual psychomotor reflexes that produce and maintain fusion are again automatic from the point of view of willed movement, with vision itself furnishing the conscious component.

There is a still higher anatomical level of control over the ocular movements. This is situated in the frontal lobe of the brain in that portion of the cortex known as the frontal eye field. This includes area 8. The usual clinical application of this area is in terms of horizontal versions. Right area 8 sends voluntary impulses down the brain stem and across the midline to set off left conjugate deviation. This again utilizes the older median longitudinal fasciculus system and enters at the level of the abducens nucleus. Thus an irritation of right area 8 will cause levoversion. On the other hand, destruction of right area 8 will result in the eyes being turned to the right. This is because left area 8 is now able to act without a balancing impulse from the right side. Vertical movements can be shown to occur experimentally by stimulation of the frontal eye field. There is no reason to doubt that the pathways for vertical movement enter at the level of the midbrain, just as do the occipital motor fibers. The pathways from area 8 are a part of the voluntary motor system and are called corticobulbar fibers. They mediate voluntary conjugate movements – *willed* movements.

The frontal eye field is also able to exert its influence on ocular rotations by controlling the psychomotor movements originating in the occipital lobe. Thus it has a double way of controlling the final nervous pathways. For an example, let us consider the voluntary act of consciously looking at an object to the right. The head and eyes turn together in that direction. Now the usual involuntary response to turning the head to the right would be twofold: first, the vestibular system would turn the eyes to the left, and the optokinetic

impulses would follow the movement of the object in fixation – also to the left. This means that in addition to voluntary movement of the eyes to the right, the frontal eye area must also inhibit, and thus exert control over, both the vestibular and fixational impulses. There is evidence to show that such inhibition by frontal cortical impulses of occipital cortical impulses actually exists for conjugate movement.

What of frontal lobe control over the vergences? Here we must enter more theoretical ground. Since convergence is an integrated part of the psychooptic reflexes from the occipital motor area, it is likely that if a "convergence center" exists, it is only in such an arrangement. A person, however, can cross his eyes willfully – but only by the voluntary act of focusing at something closer. Thus the willed act of "looking" controls the occipital convergence mechanism. It is possible also that the frontal cortical area can inhibit convergence – as it inhibits other occipital functions – and thus produce divergence. Certainly the fact that a person with accommodative esotropia can be taught to dissociate his convergence and accommodation by "seeing blurred" to maintain straight eyes is evidence of such inhibition. On this basis of reasoning, a divergence center, as such, is not necessary, and the nucleus of Perlia, the traditional convergence center, becomes merely a way station in the pathway. Experimentally, in the monkey, it is possible to elicit divergence by stimulating the appropriate portion of area 8.

It can be seen, then, that the concept of the neuroanatomy underlying ocular movement must be an active one, not merely a listing of structures concerned. Successive levels of control over the final motor pathways must be visualized, one upon the other—each capable of controlling the more primitive levels and culminating finally in the brain cortex where the site of conscious activity, the frontal cortex, is able to direct and utilize all the lower mechanisms.

THE CLINICAL NEUROLOGY OF CRANIAL NERVES III, IV, AND VI

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IT would be presumptuous to discuss the extraocular muscles and their functional movements with this group, as I am sure that everyone here has an excellent knowledge of them.

For a better understanding of how pathologic conditions affect eye movements, one should be acquainted with the basic neuroanatomy. There is much argument about this anatomy, and the absolute knowledge of it is fragmentary.

I think we could start with the cortex and follow the oculomotor nerve tracts downward to each of the centers in the brain stem and out the peripheral nerves to the eye itself, discussing the possible pathologic conditions enroute. There are two cortical areas. The one in the posterior part of the middle frontal gyrus is the voluntary center; it is known as Brodman's area 8. The other, the involuntary center, is in the occipital cortex and is most strongly represented in Brodman's area 17. We shall not discuss lesions in the occipital area because they cause visual field defects which overshadow by far the disturbance of extraocular movements.

In the voluntary or frontal center there is bilateral repression. It is for this reason that the impairment of lateral conjugate gaze that results from an acute lesion here is only transient. An example of an irritative lesion here would be a convulsive seizure which starts with lateral deviation of the eyes to the side opposite from the lesion. With an aural hallucination, the eyes deviate to the side from which the sound seems to come. With an involvement, such as acute inflammation or vascular thrombosis, in this region, there is a transient paralysis of lateral gaze to the opposite side. Their exact course and synaptic connections are not too well known, but the corticonuclear fibers from the frontal field pass through the anterior limb of the internal capsule to the separate centers in the brain stem. These individual centers have specific functions; for practical reasons we will describe only three. There are centers in the basal ganglion and superior colliculi which have to do with vertical gaze. In the lower pons there is a center on either side; these correlate horizontal gaze. Another center is in the mesencephalus, which correlates convergence. This is the nucleus of Perlia, and it lies in the midline between the motor nuclei of the third nerves.

The centers for vertical gaze, which, as we have said, are in the superior colliculus and basal ganglion structures, can be affected by lesions that paralyze them and by lesions that irritate them. An example of the results of an irritative lesion would be the oculogyric crisis. There is elevation of the eyes into the extreme position with fixation of the eyes. This is sometimes called "ceiling spasm." It is often maintained for hours. This is probably due to the basal ganglion structures.

An example of paralytic involvement of vertical gaze by a lesion in the basal

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ganglion structures would be parkinsonism. There is partial fixation, with jerky movements of the eyes as well as restriction of the movements. Parinaud's syndrome, or superior colliculus syndrome, is characterized by normal lateral excursion, absent or impaired vertical gaze with paralysis of the upward movement, and absence of the light reflex but good response to near focusing. There is ordinarily intermittent severe headache and associated nausea and vomiting caused by the sudden increase in intracranial pressure which is a result of the blockage of the flow of ventricular fluid at the origin of the aqueduct of Sylvius. This syndrome is usually due to a pinealoma, but the same syndrome occurs as a result of other lesions of the superior colliculus, including syphilitic lesions, vascular thrombosis, and infiltrative brain tumors. Tumors of the posterior cranial fossa, particularly in children, even though there is no direct involvement of the tumor with the superior colliculus, may cause Parinaud's syndrome, presumably on the basis of increased intracranial pressure from the blockage of flow of fluid into the fourth ventricle.

Next, we will take up lateral gaze and convergence. The supranuclear fibers associated with lateral gaze descend to the pontine nucleus, which lies at the junction of the pons and medulla just lateral to the nucleus of the sixth nerve. This nucleus correlates the movements of lateral gaze. Any involvement from this point upward toward the cortex would be referable to a supranuclear lesion. There is a center for convergence between the two nuclei of the third nerve which is called the nucleus of Perlia. This structure lies just ventral to the periaqueductal gray matter. There are functional connections between this point and the nucleus of the sixth nerve and the nucleus of lateral gaze. These connections make up part of what is called the longitudinal fasciculus. Involvement of the longitudinal fasciculus produces what we call internuclear ophthalmoplegia, and the symptoms vary according to the level at which involvement occurs. When the lesion involves the posterior portion of the fasciculus, there is weakness of conjugate gaze and nystagmus; the involvement is bilateral. When the lesion is extremely anterior, involving the nucleus of Perlia, there is paresis of convergence. When the lesion lies between these two points, there is muscle convergence and muscle excursion of the abducting eye. Involvement of these regions is usually by tumor, vascular lesions, and most often, multiple sclerosis.

The third nerve courses through the upper pons in a ventrolateral direction. It passes through the red nucleus and just medial to the midline and exits into the interpeduncular fossa. The nerve then takes a rather straight course to the point where it enters the cavernous sinus. Of the cranial nerves III, IV, and VI that enter at this point, nerve III, the oculomotor nerve, is the most medial.

We will now return to the third nerve nucleus and discuss the pathologic conditions which could occur along this tract and nerve. A lesion of the third nerve nucleus would give rise to a third nerve palsy and a contralateral tumor (Benedikt's syndrome). A lesion at the point at which the nerve goes into the interpeduncular fossa would cause a contralateral hemiplegia in addition to the homolateral oculomotor palsy. The usual lesion is a small vascular thrombus or a demyelinating plaque in multiple sclerosis. A lesion at the peduncle would give rise to a contralateral hemiplegia in addition to homolateral oculomotor palsy.

The trochlear nerve, or cranial nerve IV, has its nucleus just caudal to the

motor nucleus of the third nerve, and for no explainable reason the tract for this nerve courses dorsally through the pons to make its exit dorsolaterally at the junction of the pons and peduncle. It then courses ventrally around the peduncles to enter the cavernous sinus just lateral to the third nerve.

The sixth nerve has the longest course and consequently is the most vulnerable to palsy from traction, pressure by tumor, or trauma. This nerve leaves the brain stem ventrally at the junction of the pons and the medulla. Any involvement of the sixth nerve nucleus often produces a homolateral seventh nerve palsy. The nerve travels ventrally and goes beneath the lateral branches of the basilar and posterior communicating artery. It curves over the pyramid beneath the petroclinoid ligament to enter the cavernous sinus. All three nerves leave the cavernous sinus to enter the superior orbital fissures. They lie lateral to the internal carotid artery. A common lesion causing extraocular palsies in this region is aneurysm of the common carotid artery. Because of the proximity of the nerves in the cavernous sinus there is often involvement of all three nerves in the aneurysm within the sinus. If the aneurysm is just above the clinoid ligament, then the third nerve is most vulnerable. Ptosis and retroorbital pain that occurs in exacerbations may be diagnostic. The pain is from the fifth-nerve involvement. There may be involvement of all three divisions of the trigiminal nerve, or there may be only pain in and behind the eye on the same side as the lesion.

A carotid cavernous fistula may produce a pulsatile exophthalmos with a complete extraocular paralysis and also an intraocular paralysis due to involvement of the fibers from the Edinger Westphal nucleus.

The superior orbital fissure syndrome which includes paralysis of any or all the three cranial nerves, possibly with associated pain from the fifth-nerve involvement, may be the result of suppurations of the sphenoidal sinus, skull fracture, or tumor. The optic nerve may be involved.

An eighth-nerve tumor in the cerebellar pontine angle can cause a sixth-nerve palsy. Any of the nerves III, IV, and VI can be involved in toxic or inflammatory neuritis, the most frequent causes of which are diabetes, lead poisoning, and neuroencephalitis.

Of importance also is myasthenia gravis, which produces a weakness of the extraocular muscles by a metabolic defect at the myoneural junction. Neostigmine gives dramatic relief of the palsy by inactivating cholinesterase at this point.

The prognosis in cases of traumatic extraocular muscle palsy is usually good, and return of function is expected in three to nine months. This is also true of those cases of extraocular muscle palsy caused by aneurysms, unless the nerve is divided in the process of clip ligation of the aneurysm.

ANIMAL EYES

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NATURE has worked awe-inspiring miracles in adapting members of the animal kingdom to the conditions of their environment. Within the evolution of the vertebrates, we find, in general, that the number of visual elements per unit area increases. A central region in the retina develops that is highly differentiated into a fovea-centralis for high visual acuity. There is a gradual movement of the eyes forward in the head so that the visual fields overlap, thus providing binocular instead of panoramic vision. Stereopsis is made possible by decussation of the fibers of the optic nerve.

The most primitive olfactory cortex gives way to a more elaborately developed visual cortex, so that the brain is capable of estimating an increased value of complex visual designs – a stepping stone in the development of the intellectual supremacy of man.

We think of man as having the most highly developed vision of all creatures. And he probably has, as far as an all-purpose eye is concerned. Yet, in some instances, the bird in its native state has as much as eight times the central visual acuity of man.

In all probability, all protoplasmic cells have a certain susceptibility to light. Because of this sensitivity, there soon develops in multicellular forms of life a more specialized form of eye, or seeing organ, with its beginning in some pigmented cells. As the animal form becomes more complex, these pigmented cells develop into an infolding light spot with some fibrils which connect with the central nervous system and which probably evolve into the rods of the retina as the eye continues to advance in its development. This advancement continues as the fibrils form a concave area and a lens is developed within it. This final form of eye is attained in the vertebrates, and in a few invertebrates. The evolution of a species (phylogeny) is directly related to development of the original organism (ontogeny), which can be seen in the development of the human embryo. The optic vesicles, when first seen, are directly opposite one another and gradually come forward as the fetus develops. Every system and every part of man's body tells the same story. Hence, ontogeny is a condensed recapitulation of phylogeny.

The usual number of eyes in vertebrates is two, although there are some lizards which have a supplementary parietal eye. Spiders usually have eight eyes, while some of the marine worms have several hundred. Vermilia infundibulum has, on the external aspect of each bronchium, at least 200 ocelli: in all, about 11,000. The eye of the common scallop (Pecten) has its eyes in the only situation where they are likely to be of use – in a single row around the margin of the mantle. The scallop has, for a mollusk, a highly developed visual organ. Kalt describes the eyes of this mollusk as brilliant points, shining with an emerald green or purple reflection. They vary in number according to the species: 28 to 46

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on the upper half of the mantle and 15 to 36 in the lower half, measuring 0.6 mm. to 0.8 mm. in diameter. The number increases with age in the same individual.

With the exception of the lizard, Lacerta ocellata, no animal possesses a functional, useful pineal eye. The unpaired, or pineal, eye reached its highest development in the pre-tertiary period and in a number of extinct saurians—ichthyosaurus, plesiosaurus, etc.—ancestors of our birds and reptiles.

Seals are quite nearsighted on land, because the corneas of their eyes are flat and irregularly astigmatic. They neutralize this by having vertically slit pupils that immediately contract when they get out of water, giving them stenopeic vision. Water neutralizes the flat, irregular cornea, which is compensated by the eye's spherical lens. This increased power enables the seal to focus on nearby objects.

Most of the Ungulata, such as horses, gazelles, camels, and llamas, have large, oval-shaped corneas with corresponding oval-shaped pupils. This gives them a wide, horizontal field of vision. In the center of the upper border of the pupil is a deeply pigmented elevation with a corresponding protruding facet on the lower border. When light strikes the pupil, it is divided into two round pupils, one looking forward and the other looking backward. This enables the animal to secure clear, stenopeic vision for use in jumping and judging distance, while still retaining the side, horizontal field with the pupil dilated. With the eyes set prominently forward, the animal can see behind and observe the outkick of its hind legs without turning its head.

A rabbit's eyes are set so far behind that he cannot see immediately in front of him. Hunters have observed that a rabbit may run right into a man standing in a furrow down which the rabbit is speeding.

All grazing animals have very large eyes. The horse has the largest eye of any of the land mammals, and the largest eye belonging to a land vertebrate is that of the ostrich. It measures 50 mm. in diameter.

The hippopotamus, like the frog and the alligator, has its eyes as well as its nostrils elevated, so that they may be kept in the air while the rest of its body is submerged. Although this is an aquatic adaptation, it is not a visual adaptation. The hippopotamus is unique in that, although it spends most of its time in the water, it does most of its feeding on land.

Man and some of the higher apes are the only animals whose eyes show the whites to any extent. This is due to the relatively small cornea and the wide palpebral fissure which exposes the white sclera. Most animals in the wild state are farsighted, whereas most domesticated ones are prone to nearsightedness. Most animals can distinguish color, although in some this faculty is very poorly developed. Most authorities believe that fish are not able to distinguish color; they explain that the apparent ability of the fish to differentiate between colors of different flies is due to the amount of light reflected or to the intensity of the color, rather than to the color itself. Fish are extremely nearsighted in the air. Hirschberg found the refraction of the perch's eye to be between 30 and 40 diopters of myopia. He thought that in water its refraction would approximate normal and that the approach of the lens to the cornea would increase the refraction for near.

Mammals and ruminants, which run beside their mothers almost as soon as they are born have their eyelid margins separated before birth. They are born into the world with their eyes open. The young of nearly all carnivora and

of most insects and rodents spend the first weeks of their life in a prepared nest or lair and are born blind. The young of arboreal animals, such as monkeys, lemurs and the higher apes, travel about with their mothers from branch to branch immediately after birth. The baby monkeys are often carried by their mothers; the baby lemurs cling tightly to their mothers' ventral fur. Newborn primates are quite as helpless as the newborn carnivora; unlike them, however, they are born with their eyes open.

Snakes have a spectacle, or protective goggle, which is a layer of hard, transparent skin covering the cornea. The spectacle is shed when the skin is shed; thus the scratched surface is replaced with a new transparent one.

All terrestial animals have three eyelids with the exception of man and monkeys, and they have a remnant, the plica semilunaris. The third eyelid is most highly developed in birds, in which a special muscle pulls it forward. Grazing animals have a retractor muscle also, which pulls the eye backward and pushes forward the orbital fat, at the same time that the plica is pulled forward. This provides further protection against sharp blades of grass and the like.

The depth of the anterior chamber varies greatly. In fishes and amphibians the spherical lens is almost in contact with the cornea. In birds of prey the lens is widely separated from the cornea, and the depth of the chamber may be as great as 5 mm. In the largest of mammals it barely reaches 5 mm. Whereas the sclera in most mammals consists of dense fibrous tissue and is about even in thickness with that of the cornea, in fishes and birds it may contain cartilage and bone. This is generally true in birds, whose scleras contain overlapping bony plates. The whale, however, is an exception in mammals, and here we find the sclera and dural covering of the optic nerve enormously thick.

In order to obtain binocular single vision, it is necessary for the optic nerve fibers to cross. This occurs in all mammals and in most vertebrates. However, in birds and fishes, all of the fibers cross with no direct fibers to the same side. It was formerly thought that it was necessary to have partial decussation in order to have stereopsis. However, we know that birds, for example, must have perception for depth as well as for solidity; and they have complete decussation. What then, is the advantage that mammals have in possessing partial decussation? Partial decussation accomplishes the tying up of both retinas to both the left brain and the right brain centers of eye muscle control by way of the thalamus and the tegmentum, whereby what one eye is seeing is enable to control the motor impulses to both sets of eye muscles. Thus, partial decussation is not an indispensable basis for binocular single vision, but it is a logical eventual consequence of binocularity. Partial decussation has never arisen in the owl, for example, because its eyes are motionless.

The eyes of birds are large, both actually and relatively. The eyes in the swift and the owl occupy one third of the whole head; the swallow's eyes, one-twentieth of the whole body; the garter snake's eye, 1/1000 of its body. Fishes have very large eyes, especially deep sea fishes. The eye of a cod equals that of a horse in size, and that of a shark is still larger. Saurians and eels, which prefer a muddy habitat, have small and defective eyes. There is a marked disparity between the size of the brain and that of the eye in fishes and birds. Thus, the two eyes of the sparrow weigh as much as its brain. The relationship in Lophius piscatorius is tremendous. We find that large eyes

are found in animals that move the eyes or head or whole body rapidly so as to acquire distinct and instantaneous vision, e.g., birds and mammalian carnivora.

Nearly all vertebrates have six extraocular muscles; some have a seventh one, the retractor bulbi. These muscles are not always developed fully, as they are in the human eye, because in many of the birds and fishes the eye is practically motionless. In fishes, however, the function of the oblique muscles is entirely different from that in mammals. This, I am sure, is bound to be of special interest to you, as orthoptists, on account of the prevailing popularity, especially among the movie set, of the Bates system of eye care. In his book, Bates presumes to prove that accommodation for near objects is due, not to the action of the ciliary muscle on the lens in the eye, but to the action of two of the external muscles, the two obliques. He shows pictures of fish eyes with changes in the length of the eyeball supposedly caused by traction of the oblique muscles. In the first place, the photos do not show what he claims they do. Moreover, the function of the oblique muscles in the fish is entirely different from that in the higher mammals. An examination of fishes reveals that the primitive eye muscles were not developed to aim the eye at objects; rather, these reflex, or involuntary, muscles were designed to give the eyeball the balance of a gyroscopically-stabilized ship — to maintain a constancy of the visual fields despite chance buffetings and twistings of the animal's body by water currents. Fishes never keep moving objects in view by movement of their eyes. Having no neck, the fish bends or turns the whole body to face the object and keep it in the binocular field. These wheeling movements can be seen clearly in aquariums: as the fish bends its body to swim upward or downward, the eyeball makes a compensatory rotation in the plane of its equator, this movement being carried out by the action of the two oblique muscles.

Bates' contention is that the ciliary muscle, which regulates the convexity of the lens, has nothing to do with the focusing of the eye. Yet, this change in the curvature of the lens is definitely proved by observation with the slit lamp and the corneal microscope. Too, the astigmatism of the cornea can be accurately measured during both rest and accommodation with precision instruments, such as the ophthalmometer, and found to be constant. All of these measurements are made with mathematical accuracy by instruments and precise means which no scientific investigator could possibly question.

In addition to all of this, Tenon proved that during the contraction of an extraocular muscle, either by traction or by electrification, part of the aponeurosis, or capsule of Tenon, attached to the wall of the orbit, contracts so that it draws the muscle outside of the insertion, away from the globe toward the orbital insertion of Tenon's capsule, thus making no pressure on the globe.

As mentioned before, amphibians and birds have a well developed membrana nictitans, or third eyelid. They also have a retractor bulbi, which serves as a protection. This third eyelid is lubricated not by fluid from the lacrimal gland, which is poorly developed, but by the harderian gland, which secretes an oily substance that facilitates the cleansing of the eye and the removal of foreign bodies. The membrana nictitans is most highly developed in birds and in the grazing animals, in which the retractor bulbi helps to protect the eye from sharp blades of grass and the like.

In birds, a fanlike process extends forward into the vitreous from the optic nerve and almost reaches the lens. It is

very vascular and heavily pigmented. We do not know its exact function, but it probably plays a part in nourishing the vitreous and the retina. It also probably shields the retina at times from direct rays of the sun, for an eagle can take off in flight from the ground looking directly into the sun.

In fishes, there is a process running forward from the optic nerve – the falciform process with the campanula halleri at the anterior end – which corresponds to the pecten in birds and reptiles. It, however, has a different function. It extends from the nerve forward to a point just behind the iris and is attached to the inferointernal part of the equatorial border to the lens. Attached to the anterior end is the retractor lentis muscle, which pulls the lens backward during accommodation. This action is the reverse of the action in mammals, for the teleost's eye is fixed for near and accommodates for distance. in the elasmobranchs (rays and sharks), the muscle of accommodation pulls the lens, pendulum fashion, toward the cornea. In the tubular eyes of some of the deep sea fishes, the lens most likely does not move at all. In other fish, the muscle of accommodation moves the lens laterally toward the tail into the retinal cup, especially in the fish which have a fovea in the temporal periphery, where it serves for binocular vision.

In the turtle the lens is very soft and pliable. Consequently, when the powerful sphincter irides and ciliary muscles act together in compressing the anterior surface, the lens is squeezed forward through the pupil so as to make virtually an anterior lenticonus. It thus is able to accommodate both in water and in air.

Most song birds have brown eyes, whereas parrots and aquatic birds have red eyes. The color of fish eyes may change within a few hours, due to the conduction of, and alteration in, the chromophores, or red and yellow fat globules.

The pupil in practically all animals, when dilated, is round. All birds have round pupils except the owls, which have vertical slits. The horse and ruminants in general have horizontally oval pupils. There is a small minnow found off the northeast coast of South America, called the Anableps, which has both an aerial pupil and an aquatic pupil in each eye. The lens is pear-shaped with the long axis toward the aquatic pupil, which is divided from the aerial pupil by an opaque band across the middle of the cornea. As he floats near the surface of the water the minnow sees through the short axis of the lens, his eyes being partly out of the water.

Fishes are decidedly myopic, sometimes as much as 30 to 40 diopters or more, in air, although in water they are emmetropic. Birds have a special accommodative muscle, Crampton's muscle, which in contracting causes the cornea to become more convex; the lens yields to pressure of the ciliary muscle, bulges forward and becomes more convex.

Birds of prey have a very deep anterior chamber. In the sparrow, the chamber is shallow. Birds have osseous plates in the sclera between the outer and inner layers, lying anterior to the greatest circumference of the eyeball and extending almost to the edge of the cornea. The number of these ossicles varies in different species, but the average number is about 15 in most birds.

The lens is circular, or nearly so, in birds, amphibians, reptiles, and fishes. There are a few exceptions, such as the chameleon, viper, and adder. The lens is very large in fishes. The whale (not a fish) has a lens which measures 13 mm. by 16 mm.; its whole eye measures 7.5 cm. by 12 cm., which is, however, very small in proportion to the size of

its body. The lenses of fishes are hard and dry; those of birds are soft and pliable. Although the lens in the higher mammals never regenerates, the lens of a fish or rabbit will close after a traumatic injury, leaving no trace of the injury. In the salamander the lens will be entirely replaced after removal, as will the entire eye. For that matter, one can remove the eye of a small salamander and replace it with the eye of a large salamander, and it will grow and function as the original one.

In birds of prey rapid coordination between seeing and capturing their prey is necessary; therefore the optic lobes are very strongly developed. While vision for each eye is panoramic, they have binocularity and stereoscopic vision, since their second fovea is located in the temporal part of the retina. According to Casey Wood and Lindsay Johnson, wild birds are hyperopic while domesticated birds are myopic, astigmatic, or both. This was especially true of the owls which Lindsay Johnson examined in the London Zoo in 1905. Those owls which had lived in the garden for more than two years were generally less hyperopic than were those recently introduced. We know this to be true also for domestic cattle and horses when they are compared with animals of similar species in the wild state.

The Hottentots, certain natives of India, and the Terra del Fuegans, are known to have phenomenal visual acuity. Their sharpness of sight is due, according to Casey Wood, to the slenderness of their retinal cones.

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HISTORY OF ORTHOPTICS AT MASSACHUSETTS EYE AND EAR INFIRMARY

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MY interest in orthoptics goes back a good number of years. A long time ago I was one of a group of ophthalmologists who met in Washington, D. C., and formed the American Orthoptic Council. The purpose of this Council, as you know, was to conduct examinations, to certify successful candidates, and to lay down a code of ethics by which we hoped to steer orthoptics in the proper direction. At that time your profession had fallen into some disrepute because of the extravagant claims made by some orthoptists and some ophthalmologists who felt that all cases of squint could be straightened by nonsurgical means. Ever since Javal, in 1865, laid down the modern concepts of orthoptics, there have been two schools of thought regarding it: one, which felt it was a cure-all for almost all disturbances of ocular motility, and the other, which regarded it as "damn nonsense." As in many controversies, the truth lies somewhere between the two extremes. Orthoptics managed to survive the storm and, in my opinion, is here to stay because of the great help it has been to ophthalmologists in diagnosing and supervising their squint cases. Our clinic here, under Miss Stromberg, has been exceedingly busy, and all members of the staff appreciate the help it has offered them.

In 1937, I presented a paper on orthoptics before the New England Ophthalmological Society. I would like to quote from the first paragraph of that paper:

> The purpose of this short talk is to acquaint you with the latest developments in orthoptic training and at the end of it to give the results in a short series of my own cases. I am not going into a long disussion as to the nature of binocular vision and abnormal retinal correspondence, preferring to leave these subjects to Dr. Verhoeff, whose paper will deal with them in a much more scientific manner than is possible for me to do.

Now, nineteen years later, history repeats itself; again Dr. Verhoeff is following me, only this time I have taken the precaution not to say anything scientific – merely introductory.

In that paper of mine, I presented a number of cases in which I had been able, by means of orthoptics, to decrease the squint greatly. The successful ones were of children with accommodative squint whose eyes had been straight while they were wearing their glasses. I presented pictures of the patients before and after treatment and showed how these children, after treatment, could hold their eyes straight without benefit of glasses. In discussing my paper, Dr. Verhoeff said he could not understand what this was all about. After all, the patients' eyes were straight while they were wearing their glasses anyway, and all I had done was to make it possible for them to hold their eyes straight without glasses, which he did not think was such a

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great accomplishment because all they had to do to have straight eyes was to wear their glasses. This rather deflated me because I was rather proud of the results; but Dr. Regan of the Boston City Hospital came to my aid by saying, "Dr. Verhoeff, this may not seem like anything to you but, believe me, it makes a lot of difference to a young bride." Dr. Verhoeff allowed that he had not thought much about this particular aspect of the situation.

I would now like to introduce to you Dr. Frederick Verhoeff, who for years, has had such a wonderful influence on ophthalmology here and everywhere. He has made original contributions to binocular vision, has devised a test for stereoscopic vision which, to my mind, is the best, and, by his great intellectual honesty, has prevented us from becoming overenthusiastic about various procedures until the facts have been proved.

NEW ORTHOPTIC TECHNIQUES

FRANCES FOWLER, O.T.

LOS ANGELES, CALIFORNIA

MY subject is new orthoptic techniques. You, as ophthalmologists, and I, as an orthoptist, know that orthoptics is an adjunct to ophthalmology, and that new orthoptic techniques will depend largely upon new ophthalmological research.

In this paper will be demonstrated new therapeutic methods developed from the use of peripheral fusion and peripheral stereopsis.

In a moment I will discuss the practical aspects, but first I would like to tell you how thrilled I was to read the account of Linksz, telling of the dramatic effect on himself of Burian's experiment indicating the strength of peripheral fusion.[4] The peripheral stimuli, which were strong enough to break central fusion, were but vaguely perceived by the subject; their shape was not even recognizable, yet the binocularly seen central image would appear double, no matter how hard the observer tried to maintain central fusion.

The role of peripheral fusion has been emphasized by Winkelman,[7] Keiner,[3] Ogle[5] and others as well as Burian.[2] Winkelman,[7] modifying Burian's experiment, further demonstrated that even non-identical images, which, if superimposed, would cause retinal rivalry but not sensory fusion, imaged on disparate points of the retina, induced a fusional movement of the eyes to bring the images on corresponding points. Keiner[3] specifically calls the arousing of peripheral fusion the provisional aim of orthoptics; only after the training of the peripheral retina can one proceed to the training of simultaneous binocular vision with the retinal center, the final aim of which is central fusion. Burian[2] says that by concentrating almost exclusively on central retinal areas in training binocular vision, a powerful tool is being neglected.

In patients with a central sensory disturbance, such as suppression, or anomalous retinal correspondence, or diplopia, there may nevertheless be a strong peripheral fusion ability already present, permitting extensive fusional movements. Furthermore, Burian maintains that *only* in patients who have such a sensorial disturbance of the central parts of the retinas are fusional movements elicited by peripheral retinal stimuli, and if no central disturbance exists, the patient suppresses the image of one retinal periphery.[1]

In practice, this involves the simple principle of starting with the skill the patient already possesses, and from there working gradually into the field where his disturbance lies. In these patients with a central sensory disturbance we therefore start in the periphery by using peripheral fusion slides, or peripheral stereopsis slides, obtain our fusional amplitudes, and finally eliminate central suppression with the conventional first grade slide.

In Burian's original experiments, and in later experiments by Ogle and oth-

Presented at the Pacific Coast Oto-Ophthalmological Society, Phoenix, Arizona, April 1956.
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ers, peripheral fusion was demonstrable at as much as twenty degrees from the fixation point, if visual acuity was exceptionally good, but most of the experiments were with peripheral targets at five degrees, and at twelve degrees from the fovea.[5] The synoptophore provides an angle of slightly over thirteen degrees from the fixation point, the troposcope provides an angle of ten degrees horizontally, and eleven degrees vertically, so that from the point of view of providing an adequate visual field, a major amblyoscope is a satisfactory practical therapeutic apparatus.

To illustrate this more directly, I will proceed with specific categories.

First, in amblyopia ex anopsia, when there is no strabismus present, the eyes can be presumed to have remained "straight," because of some peripheral fusion hold. These children, with visual acuity in one eye as low as 20/200, and total suppression of one central image, nevertheless have excellent recognition of stereopsis, and excellent fusional amplitude, provided targets are used large enough to avoid the central, suppressing area. With these children in the past, the usual procedure of total occlusion of the non-amblyopic eye has at least occasionally, and perhaps frequently, led to the onset of strabismus which required surgery to correct. Many doctors have felt that the decision between allowing the amblyopia to remain, or creating a strabismus by occlusion, was a difficult one. By utilizing peripheral fusion, fusional amplitudes can be increased immediately from the beginning of the occlusion period. The first indication of esophoria or exophoria can be counteracted immediately with fusional amplitude in the appropriate direction, and thus a possible strabismus prevented.

Secondly, in cases of eccentric fixation, when the normally fixating eye is covered, and the deviated eye takes up a definite eccentric fixation: whether there is a macular lesion, or whether the macula has been inhibited so strongly that an eccentric point is chosen for fixation, the result is that occlusion of the good eye will have no effect in improving macular vision of the deviating eye. We have succeeded in establishing a form of stable binocular vision with peripheral fusion, in spite of the low acuity of one eye. We begin

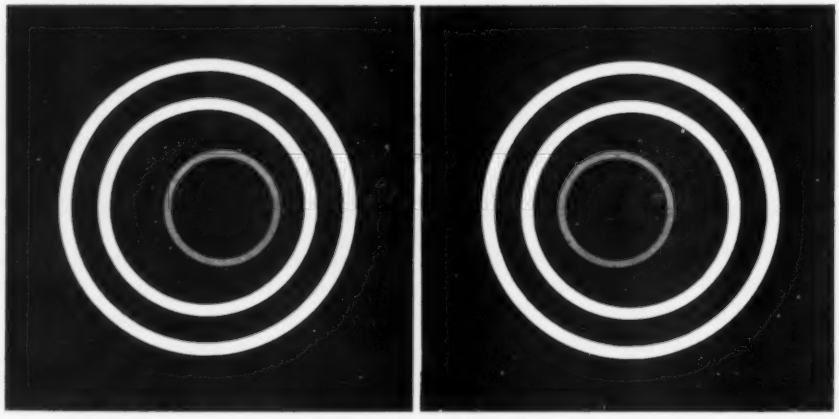

FIG. 1 — Inner ring is red.

by occlusion of the eccentrically fixating eye for a period long enough that the fixation of the amblyopic eye appears no longer eccentric but merely wandering. This has taken as little as three months, and as long as one year. At this time orthoptic treatment is started, with large stereoscopic rings (fig. 1). As soon as there is some fusion demonstrated, the patient is immediately returned to the ophthalmologist for consideration of surgery. If there is no fusion response in twelve treatments, treatment is resumed immediately postoperatively.

SLIDES

Swan has used, in similar cases, a large green ring with a central dot for fixation presented to the normal eye, while a red ring with a blank background is presented to the amblyopic eye.[6] The rings appear gray or black when fused, but bright red or green when seen separately.

Working as I do with young children, I have had more success with the stereoscopic targets shown where the effect is not lessened by momentary change of fixation, or even momentary failure of attention.

I want to show you how a slide developed quite theoretically proved impractical in actual use. In case after case I found the peripheral stereoscopic ring targets almost immediately effective in strabismus with or without normal retinal correspondence, eliciting prompt and correct stereopsis, and fusional movements when the tubes of the amblyoscope were converged and diverged from the patient's angle. However, it is generally accepted that evaluation of a child's fusion status cannot be done properly without steady and accurate fixation for at least several seconds. I thought, therefore, that this slide could be modified and improved by adding a fixation object in the center, of interest to a small child, and small enough to elicit steady foveal fixation. However, it did not prove to be useful. When the fixation object was fixated steadily, the stereopsis was markedly reduced. When the picture was gazed at as a whole, the stereopsis increased, but when the outer ring was fixated at any point on its circumference, the inner, disparate rings then falling on the periphery, the stereopsis was far more noticeable. And in any case, wandering gaze over the entire picture elicited the stereopsis, and increased the amount of fusional amplitude obtainable, when the central fixation object was eliminated.

Thirdly, in anomalous retinal correspondence, orthoptic treatment has in the past been prolonged, because we first tried to obtain simultaneous macular perception using dissimilar slides, as with the lion and cage on the major amblyoscope. About 1945, Marianne Eyles and I began to notice during our experiments, that patients with anomalous correspondence obtained stereopsis and fusion long before they could obtain simultaneous macular perception with dissimilar slides. A series of slides using a peripheral fusion element, combined with various sizes of central simultaneous perception elements, was devised, and has consistently proven to shorten the period required to establish normal correspondence.

The fusion elements of these slides (figs. 2, 3, 4) may be fused at the first trial; more often they are overlapping at first, then fused. The dissimilar portions of the slides may be seen superimposed immediately when surrounded by the fusion element, even though they are never seen superimposed without the fusion element; sometimes they remain in crossed diplopia with gradually decreasing separation for several treatments.

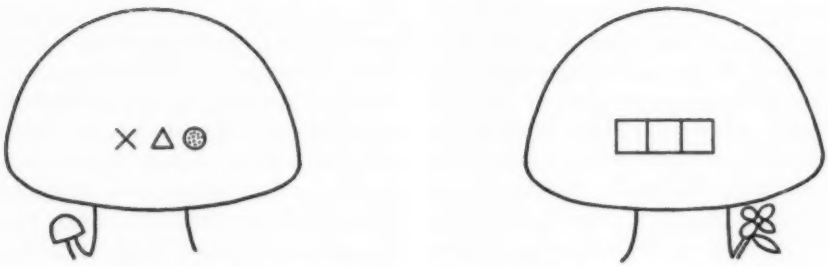

FIG. 2 – Peripheral fusion. Foveal S.P. 1°-3°.

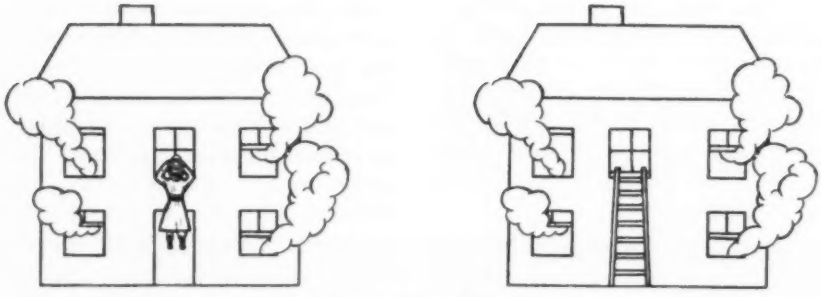

FIG. 3 – Peripheral fusion. Macular S.P. 3°-5°.

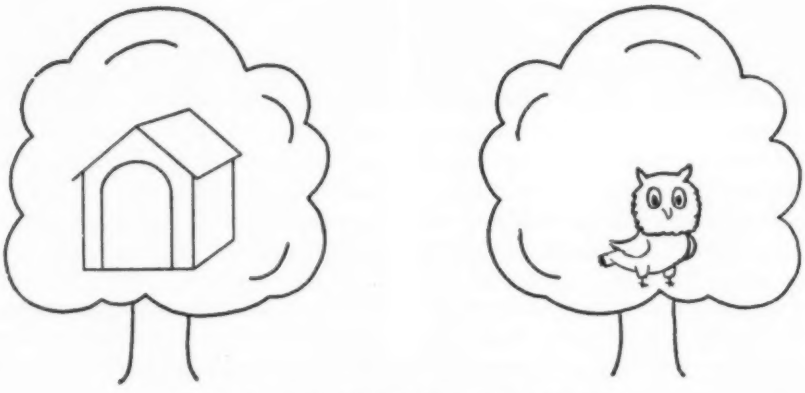

FIG. 4 – Peripheral fusion. Paramacular S.P. 5°-8°.

We have added a motility factor, finding that rapid movement of the target at varying speeds and varying amounts of sweep, would elicit fusion, where an unmoving target continued to be seen as diplopia. The fusion elements of the peripheral slides become overlapping, then fused, first with rapid oscillation and wide sweep of the targets, then with a gradually decreasing speed and sweep of oscillation. When the dissimilar targets are seen superimposed with movement, then ordinary dissimilar slides, as the lion and cage, are quickly substituted, because at this stage, the anomalous correspondence is corrected. Finally, the movement is reduced to zero, while superimposition is maintained. Concurrently with this procedure, fusional amplitude can be developed in each direction even before the dissimilar elements are seen superimposed.

I am not discussing any of these forms of treatment as apart from surgery; all these methods are pre- or postoperative. Most postoperative deviations occurring after what first appeared to be a good cosmetic result, are due to absence of fusion hold, in its turn due to uncorrected anomalous retinal correspondence, monocular or alternating suppression. Even moderately efficient peripheral fusion hold will stabilize each type of case in its excellent postoperative position, allowing the remaining central disturbances to be eliminated postoperatively.

My last category concerns the heterophoria cases, especially convergence insufficiency. Nothing but good fusional amplitude with foveal targets is as a rule sufficient to relieve the symptoms of discomfort of patients with convergence insufficiency. Yet many patients, treatment after treatment, are unable to maintain fusion convergence for more than ten to fifteen diopters, where the normal is seventy to eighty diopters. This can be true where convergence to a light or pencil is full; indeed Scobee[8] defined convergence insufficiency not necessarily as a deficiency in the convergence near the point, but as a deficiency of fusion convergence, with accommodation more or less constant, as with base out prism added while fixating print at a fixed distance, or on a major amblyoscope. This block at ten to fifteen diopters base out on the major amblyoscope can be overcome by presenting the patient with the large peripheral rings which employ peripheral fusion and peripheral stereopsis. Only then will he experience the unfamiliar, and possible uncomfortable feeling of the first convergence to thirty or forty diopters. One can then continue with the peripheral slides for perhaps six successful efforts, then reinsert first the paramacular, then macular, then foveal fusion slides in quick succession. A marked increase in convergence may then be accomplished in one visit, and the succeeding visits spent on building up the full range with foveal targets, which procedure is almost always necessary for thorough relief of symptoms.

In summary, the use of peripheral fusion slides and peripheral stereoscopic slides has been evolved into new orthoptic techniques resulting in quicker orthoptic results. Targets and techniques are demonstrated using peripheral fusion and peripheral stereopsis, in cases of amblyopia ex anopsia with straight eyes, in eccentric fixation, in anomalous retinal correspondence, in pre- and postoperative orthoptics, and in convergence insufficiency.

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DISCUSSION

JAY BUTLER, M.D., Portland, Oregon: Miss Fowler is to be complimented on presenting very clearly the most important improvements in orthoptic technique in the last few years. We are thoroughly in accord with her views as to the type of target and the importance of developing peripheral fusion. Orthoptics is a field that lends itself to abuses, and this has led some men into the trap of discontinuing it entirely. It does, however, have an important place in the treatment of strabismus, and if properly utilized can be of great help. The establishment of peripheral fusion will many times save what would otherwise be a surgical failure. With certain reservation, we favor early surgery. In children who are old enough for orthoptic training when first seen, preoperative and postoperative orthoptics is our usual form of treatmeant. The techniques as described by Miss Fowler we use with variations in our own office, but in general they are based on the same principles. We would like to point out there are only a limited number of procedures that can be used in the treatment of strabismus: namely surgery, glasses, patching, orthoptics and psychiatric care. We believe the best results can be obtained in the treatment of strabismus when all available methods are properly utilized. In the older group, the orthoptist can be a big help in picking up psychiatric difficulties and stimulating the interest of the child in trying to develop binocularity.

OCULAR TORTICOLLIS

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OCULAR torticollis may be defined as an abnormal position of the head caused by a disturbance of ocular motility, usually involving one or more of the vertically acting muscles. The condition has been termed variously as head tipping, head tilting, abnormal head posture, or corrected posture. Although occasionally present in cyclophoria, an abnormal position of the head more frequently is the predominating feature of squint, both concomitant and noncomitant, especially in children. Its presence is always of great significance in the consideration of surgical treatment of ocular muscles.

Head tilting has been the subject of confusion in ophthalmic literature and is often the cause of difficulty in the diagnosis of oculomotor disturbances. Also, teaching of the subject has been inadequate at best. If for no more valid reasons, these factors are sufficiently important to make the subject of interest to the orthoptic technician, even though the condition is based more on motor than on sensory findings.

Abnormal head posture may be observed frequently in examination of patients with an ocular muscle disturbance, be it vertical or torsional phoria or a concomitant or nonconcomitant tropia. For instance, the effort to overcome an unnoticed hyperphoria may enhance the subsidiary action of both the vertical recti and the obliques, resulting in cyclophoria. And, conversely, a vertical or torsional deviation of congenital origin, possibly due to a primitive weakness of a vertically acting muscle, usually the superior rectus, may lay the basis for the development of a convergent squint.

In paretic cases of congenital origin, the patient is usually not aware of his abnormal head position. This may easily escape the notice of the examiner and, especially, the parents who are accustomed to seeing the patient with his "usual" posture. The altered head position is made more elusive because it is generally accompanied by scoliosis involving a compensatory curvature of the cervical, thoracic, and lumbar vertebrae, and sometimes a slight tipping of the pelvis. It is adopted in compensation in the absence of obtrusive diplopia (alternation). When the paralytic squint is of acquired origin, the patient is quite aware of his head posture and assumes the abnormal position in an attempt to overcome diplopia; there are no organic changes in the structure of the vertebrae or in their muscular attachments. Head tilting is occasionally seen in comitant squint in older patients, the squint probably having been of a paralytic nature during early infancy. On the other hand, the head-tilt in such cases may be eliminated by the development of suppression.

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On clinical analysis, the various rotations of the head of ocular origin can be subdivided into several components around the three principal axes: vertical, transverse, and anterior-posterior. Diplopia, the most annoying symptom of paralytic squint, by its nature will determine in large part the type of abnormal head posture. Horizontal diplopia, homonymous or heteronymous, may be rendered less annoying by turning the head to one side or the other. Vertical diplopia may be decreased by elevation or depression of the chin. Torsional diplopia can be minimized by tipping the head toward one shoulder, because of the tendency of the vertical corneal meridians to remain vertical and parallel by the process of normal conjugate cycloversions (fig. 1).

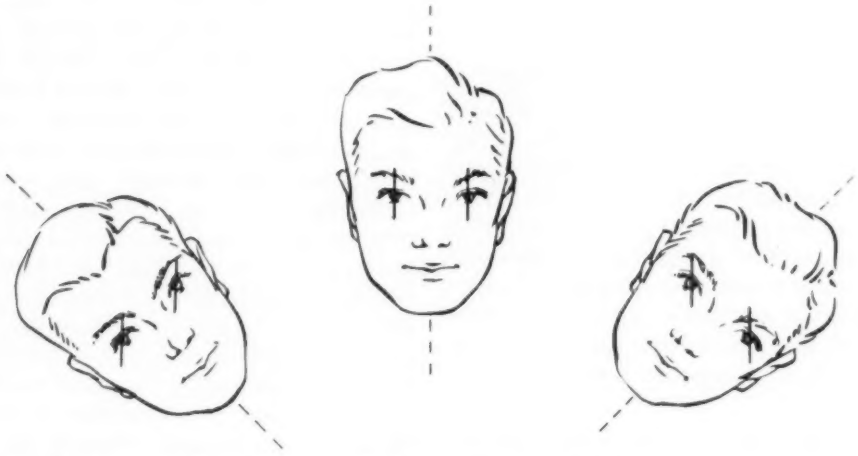

FIG. 1 — Normal conjugate cycloversions when head is tipped to right and left.

PARALYSIS OF THE HORIZONTALLY ACTING MUSCLES

The simplest situation is one involving paresis of the horizontally acting muscles. In right lateral rectus paresis (fig. 2), for example, the right eye is adducted and the patient experiences uncrossed horizontal diplopia, with the greatest separation of the images occurring in right lateral gaze. If the face be turned to the right and the eyes remain fixed, because of the optic fixation reflexes, the result is equivalent to turning the eyes to the left, where diplopia is decreased or absent. The reverse situation is found in the presence of right medial rectus paresis (fig. 3).

Hence, in the presence of a paresis of a horizontally acting muscle, the face is turned toward the field of action of the paretic muscle. Turning the face to the right, therefore, is suggestive of a weakness of a right hand rotator, that is, the right lateral rectus or the left medial rectus, whereas turning the face to the left suggests weakness of either the right medial rectus or the left lateral rectus.

Long-standing paralysis of a horizontally acting muscle may be accompanied by contracture of the direct antagonist; and in this event the mere rotation of

the face will no longer completely eliminate horizontal diplopia. However, because raising of the eyes favors divergence and lowering of the eyes favors convergence, elevation or depression of the chin may be adopted in this circumstance. In the example just mentioned of right lateral rectus palsy with the face turned to the right, esotropia may be further reduced by depressing the chin (fig. 4); and, in the case of right medial rectus palsy the exotropia is lessened by turning the face to the left, and may be further decreased by elevating the chin (fig. 5).

PARALYSIS OF THE VERTICALLY ACTING MUSCLES

A more complicated problem is presented by postural changes encountered in relation to the vertically acting muscles. These muscles have component actions in more than one direction; they act as elevators, as depressors, and as horizontal rotators, and they also have a torsional capacity (the principal action of the obliques).

In general, it may be said that in cases of paralysis of an elevator, the chin is elevated, whereas in those of

FIG. 2 – Right lateral rectus paresis. Face turned to the right.

FIG. 3 – Right medial rectus paresis. Face turned to the left.

FIG. 4 – Right lateral rectus paresis. Face turned to right, chin depressed.

FIG. 5 – Right medial rectus paresis. Face turned to right, chin elevated.

a paresis of a depressor, the chin is lowered.

Paralysis of the Superior Rectus

Palsy of the right superior rectus – the muscle which normally produces elevation, adduction, and intorsion – will allow the eye to be hypotropic and abducted, with the corneal meridian tipped outward. The false image, as the patient sees it, is displaced upward, inward (crossed), and inclined toward the normal, or left, side. These are the findings when the left, or nonparalyzed, eye is used for fixation. Because the superior rectus is a maximum righthand elevator, the patient by elevating the chin will depress the eyes in the orbit, and by turning the face to the right will, in effect, turn the eyes to the left, or opposite, side (fig. 6). As a result, horizontal and vertical diplopia may be reduced by turning the face toward the direction of the maximum vertical pull of the paralyzed muscle. This rule, stressed in most textbooks, is not infallible, unfortunately, for the horizontal deviation (exotropia) in palsy of a vertical rectus may be very slight or modified by a pre-existing esophoria or exophoria, that is, the face may be turned to the left (fig. 7). Moreover, elevation of the chin may not be required if the patient learns that by merely turning his head, his paralytic eye is placed in abduction and out of

FIG 6 – Right superior rectus paresis, primary deviation. Face turned to the right, chin elevated, head tipped to left.

FIG. 7 – Right superior rectus paresis, primary deviation, with pre-existing esophoria. Face turned to left, chin elevated, head tipped to left.

FIG. 8 – Right superior rectus paresis, with extortion of right eye. On forcibly tipping the head to the right, contraction of the right superior oblique overcomes extortion, but also produces slight depression of right eye.

the field of maximum vertical action of the paretic muscle. The torsional diplopia experienced in palsy of the right superior rectus muscle, due to extorsion of the right eye, can be decreased by tipping the head to the left, or opposite, shoulder, that is, by the artificial production of right conjugate cycloduction of the two eyes. If the head is forcibly tipped to the ipsilateral, or right, shoulder, the right extorsion may be partially or completely overcome by contraction of the ipsilateral, or right, superior oblique, which at the same time will cause depression of the right eye, since this vertical action is unopposed by the paretic superior rectus (fig. 8). This is the basis of the Bielschowsky test for differentiating between a paresis of the superior rectus and a paresis of the contralateral superior oblique.

The tipping of the head to the opposite shoulder, as just mentioned, is not a constant finding in a superior rectus palsy as modified by two circumstances. When the head is tilted to either shoulder, the ipsilateral, or lower, eye is raised slightly (its image is lowered), and at the same time the contralateral, or higher, eye is lowered (its image is raised). Hence, in right superior rectus paresis, in which the right eye is hypotropic, if the vertical diplopia is greater than the torsional, the patient may experience relief by tipping the head to the right, or paralyzed, side, thus raising the paretic eye (fig. 9). This is contrary to the above-mentioned rule and is encountered not infrequently in cases of congenital superior rectus paresis.

The common finding of tipping of the head to the opposite, or normal, side in superior rectus palsy may also be altered by secondary deviation. For example, in right superior rectus paresis of long standing the right eye may fix, and contracture of the contralateral yoke, or synergistic, muscle, the left inferior oblique, may dominate the picture. The left inferior oblique normally causes an outward deviation of the vertical corneal meridian and, if overacting, will produce marked left extorsion. Inclination of the head to the side opposite to the overacting inferior oblique, in this case to the right (fig. 10), will lessen torsional diplopia by producing relative left conjugate cycloduction of the two eyes.

Thus, depending on the amount and type of diplopia and on the fixing eye, considerable variation in head posture

FIG 9. — Right superior rectus paresis. Right or hypotropic eye is raised (its image is lowered) by tipping head to right.

FIG. 10 – Right superior rectus paresis, secondary deviation. Face turned to right, chin elevated, head tipped to right.

intorsion, when paretic will allow the eye to be elevated, adducted, and extorted. Since torsion is the most important action of the muscle, torsional diplopia will be the predominating symptom in its paralysis. This symptom is most annoying, since the images are seldom sufficiently separated, one from the other, to permit easy suppression. In a case of paresis of the right superior oblique, for example, the extorsion of the right eye can become lessened by tipping the head toward the left, or contralateral, shoulder (fig. 11), thus producing relative right conjugate cycloduction of the two eyes. Moreover, the vertical separation of the images, resulting from right hypertropia, is lessened by depressing the chin or relatively raising the eyes in the orbit. This carries the eyes out of the field of maximum vertical action of the paretic mus-

may be found in the presence of superior rectus paresis. In primary deviation, that is, with the nonparalytic eye fixing, the chin is up, the face is turned to the ipsilateral, or paralytic, side, and the head is tipped to the contralateral shoulder. On the other hand, if the paralytic eye is made to fix, the chin is raised, the face is turned to the paralytic side, and the head is tipped to the shoulder of the paralytic side. When ocular torticollis is the predominating feature, this latter condition usually prevails, probably because it permits wider separation of the double images, facilitating suppression.

Paralysis of the Superior Oblique

The superior oblique, which normally would cause depression, abduction, and

FIG. 11 – Right superior oblique paresis, primary deviation. Head tipped to left, face turned to left, chin depressed.

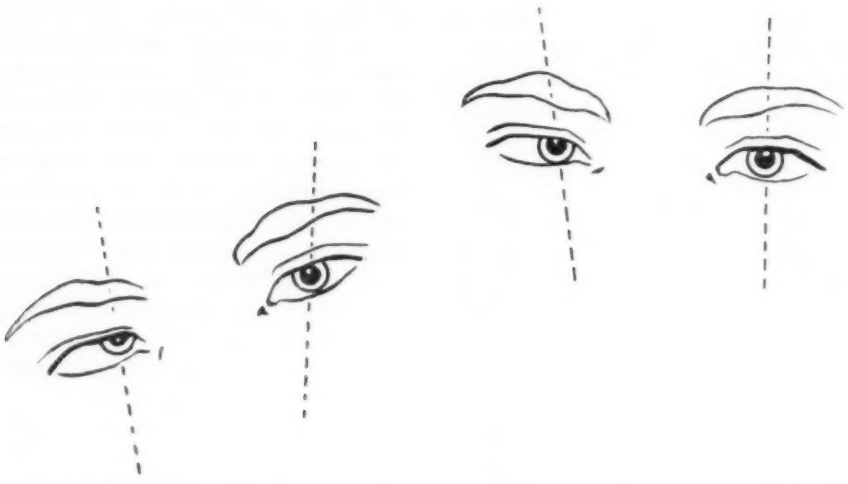

FIG. 12 – Right superior oblique paresis, with extortion of right eye. On forcibly tipping the head to the right, contraction of the right superior rectus overcomes extortion but also produces some elevation of right eye.

cle, that is, down and in (which is especially annoying in reading). Furthermore, the horizontal, or crossed, diplopia due to the exotropic component of the deviation, is decreased by turning the face to the left, or normal, side, resulting in relative right lateral version of the eyes. Thus, tipping of the head to the uninvolved side is a characteristic posture in paralysis of the superior oblique. However, if the head be forcibly tipped to the side of the paralyzed superior oblique (fig. 12), the ipsilateral superior rectus may produce elevation of the ipsilateral eye (as in the Bielschowsky test) in an attempt to overcome extorsion due to the paralysis. In contrast, if an eye with a paretic superior oblique is used for fixation, that is, secondary deviation, tipping of the head to the paralytic side, is generally encountered (fig. 13). For example, if the right superior oblique is paralyzed, and the right eye is used for fixation, overaction of the contralateral yoke muscle, the left inferior rectus, will be produced. This results in extor-

FIG. 13 – Right superior oblique paresis, secondary deviation. Head tipped to right, face turned to left, chin depressed.

sion of the left eye which can be overcome by tipping the head to the right shoulder, that is, by placing the eyes in left, conjugate cyclodeviation.

PARALYSIS OF OTHER MUSCLES

In like manner, primary paralysis of the right inferior rectus muscle may be found to produce a head-tilt to the normal side (left) with turning of the face to the affected side (right) with depression of the chin (fig. 14). The head tilting in this case is probably due more to the torsional diplopia caused by the overacting antagonist, the ipsilateral superior oblique, than to the effects of the paretic inferior rectus.

FIG. 15 – Right inferior oblique paresis. Head tilted to right, face turned to left, chin elevated.

FIG. 14 – Right inferior rectus paresis. Head tilted to left, face turned to right, chin depressed. (The head tilting in this case is usually due to overaction of the left superior oblique.)

In contrast, paralysis of the inferior oblique, which is extremely rare, is accompanied by tilting of the head to the ipsilateral side, elevation of the chin and turning of the face to the normal side (fig. 15). Reference has already been made to the overaction of the inferior oblique, which is encountered far more frequently and is of greater importance than paralysis of the muscle.

When multiple paralyses are encountered, that is, simultaneous involvement of more than one muscle in either one or both eyes, the patient chooses that position which allows binocular single vision with the greatest relief to the paralyzed muscles. In these cases, analysis of the head posture is of little practical help.

DIFFERENTIATION BETWEEN OCULAR AND ORDINARY TORTICOLLIS

Confusion has often arisen between a vicarious head posture due to an abnormality of the ocular muscles and that resulting from contracture of the sternocleidomastoid muscle, often referred to as ordinary or orthopedic torticollis (fig. 16). The latter is characterized by a very obvious head-tilt, facial asymmetry, gross contracture of the sternocleidomastoid muscle on the side of the neck to which the head is tipped, inability to straighten the head, and elevation of the chin. The face is turned contralaterally to the head-tilt, and the ocular rotations and muscle balance are normal. In torticollis of ocular origin, the head can be straightened, since there is no cervical disorder, but in this act, diplopia or suppression is always elicited, together with normal conjugate movements and an abnormal muscle balance.

The mechanism of the vestibular reflexes, the disturbance of which is thought to be responsible for abnormal head posture in oculomotor paralysis, is fairly well understood. However, many cases of paralysis do not fit into the accepted scheme. For example, occlusion of a paralyzed eye invariably causes head-tilt to vanish, but the closing of the sound eye produces no such effect. In contrast to this, if the paralytic eye becomes highly amblyopic and torsional diplopia is no longer experienced, the pre-existing head-tilt still persists. Also, the presence of head-tilt not accompanied by binocular fixation in the central field invariably indicates that binocular vision formerly had been present, and may be regained if appropriate surgical measures are applied.

FIG. 16 – Right: ordinary, or orthopedic, torticollis. Left: ocular torticollis.

In general, it can be said that abnormal postural positions are characteristic of various oculomotor deviations. Elevation or depression of the chin is a constant finding in paresis of a vertically acting muscle, and occasionally in the case of a horizontal deviation. Rotation of the face usually accompanies a horizontal disturbance but, because of its variability, cannot be considered as a reliable finding. Tipping of the head is diagnostic of a torsional defect; it is dependent on the development of fusion and binocular single vision. Postural changes, therefore, may be regarded as an adjunct in the diagnosis of oculomotor disturbances, but not as a substitute for time-tested methods. Ocular torticollis does not fit into stereotyped patterns, or follow rules of texts, although there are usually very good reasons for each particular head posture.

A DISCUSSION OF ANOMALOUS RETINAL CORRESPONDENCE

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THE conception of the diagnosis and treatment of anomalous retinal correspondence varies widely. Its incidence in cases of strabismus is reported by some as 45 per cent and higher, while others deny that it occurs. Adler, Burian, and Swan have contributed greatly toward consolidating this much confused issue by pointing out factors of a definite physiologic and anatomic nature. Their understanding of the neurophysiologic process of anomalous correspondence gives this subjective phenomenon a working, objective foundation. From Burian's works the following definition is quoted: "In anomalous correspondence the two foveas which are normally corresponding points lose their common visual direction and acquire two different visual directions." When the foveas have a common visual direction, the correspondence is said to be normal. In such a case, esotropia will result in homonymous diplopia (fig. 1).

Anomalous retinal correspondence is present when an off-foveal area in one eye assumes the same visual direction as that of the fovea in the other eye (fig. 2). This may result in a blending of the images seen by each eye or only in a crossing of these images at a lesser angle of deviation (the subjective angle) than the angle of deviation found upon examination (the objective angle). The difference between these two angles is the *angle of anomaly*. An anomalous correspondence is demonstrated on the major amblyoscope by means of the corneal reflections, the cover test, and the cover-uncover method.

The examiner uses the cover test to determine the objective angle of deviation. This is accomplished on the major amblyoscope by turning off the light before one eye and allowing the other eye to fixate the picture target. This process is continued, using each eye alternately, as the arms of the major amblyoscope are moved toward the objective angle of deviation until all movement of the eyes ceases. At this point, bifoveal fixation should be present, provided that visual acuity is adequate and/or suppression is not too strong.

At the subjective angle of deviation the cover-uncover method, as well as the cover test, is used by the examiner. This method differs from the cover test in that an interval is allowed during which both eyes remain uncovered. During this interval when both eyes are uncovered at the subjective angle, there may be a movement of the eyes to recover fusion, when fusion is possible. It is well to keep in mind that the subjective angle may not be the same as the objective angle, and that a difference in these two angles does not alone

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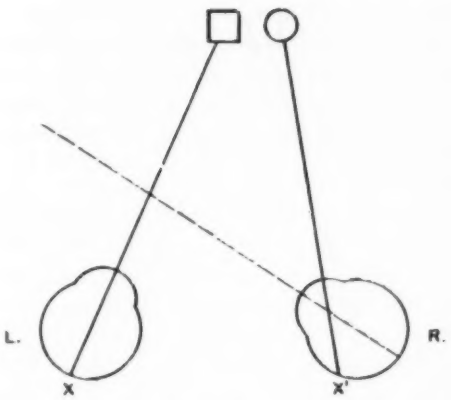

FIG. 1 – Homonymous diplopia resulting from right esotropia in a subject with normal retinal correspondence. The image falling on the left fovea (X) is projected straight ahead; the image falling on an off-foveal area of the right retina (X') is projected to the right.

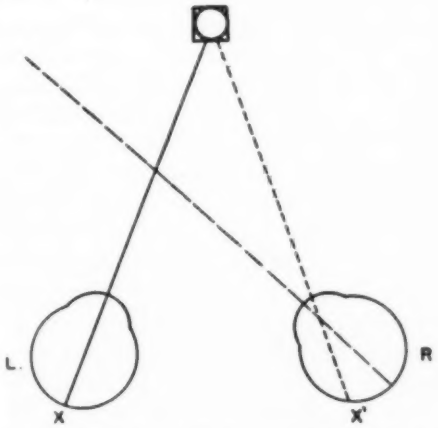

FIG. 2 – Anomalous retinal correspondence in right esotropia. The image falling on the left fovea (X) is projected straight ahead; the image falling on an off-foveal area of the right retina (X') is projected to the same point, instead of to the right as in figure 1. X and X' now have the same visual direction, although they are disparate retinal elements.

constitute an anomalous retinal correspondence.

In testing retinal correspondence at the subjective angle, the examiner's choice of the eye to be covered is governed to a great extent by the position of the corneal reflections. In esotropia with anomalous correspondence, at the subjective angle the corneal reflection in the nonfixating (deviated) eye will be seen on the temporal side of the cornea, and in the fixating eye, centered on the cornea slightly to the nasal side. Upon covering the fixating eye, the deviated eye usually will make a movement outward to fixate if foveal visual acuity is present, thereby using a visual direction different from the one habitually used. If amblyopia is present, the deviated eye may or may not move, depending upon the degree of amblyopia and the size of the target used. When the cover is removed from the preferred fixating eye, this eye usually resumes fixation and the nonpreferred fixating (habitually deviating) eye makes a movement inward. In the latter eye foveal suppression occurs, and an off-foveal area becomes associated with the fovea of the fixating eye, establishing anomalous retinal correspondence.

Anomalous correspondence is corrected when the patient learns to use a new visual direction in the deviated eye that corresponds to the visual direction in the other eye. This learning process usually can be aided by orthoptic training. The examiner tells the patient where the image of the picture target should be located when both eyes are uncovered. The most direct controlled method that can be employed is desirable. We require the patient to maintain bifoveal fixation at the objective angle of deviation and/or to change fixation from the habitually fixating eye to the habitually nonfixating

eye at the subjective angle. In certain cases this produces monocular diplopia; sometimes it occurs spontaneously at the objective angle, and sometimes it is elicited at the subjective angle of deviation. It may be fleeting or prolonged. At the time the habitually nonfixating eye changes its visual direction, the examiner will notice a movement of the eye. With a new localization of the visual direction and awareness of the old one still present, the fovea now has a two-directional projection and monocular diplopia (binocular triplopia) results (fig. 3). In alternating esotropia binocular tetralopia may occur (fig. 4). When monocular diplopia occurs at the subjective angle, the new visual direction is established at this angle before progress is continued through the angle of anomaly to the objective angle.

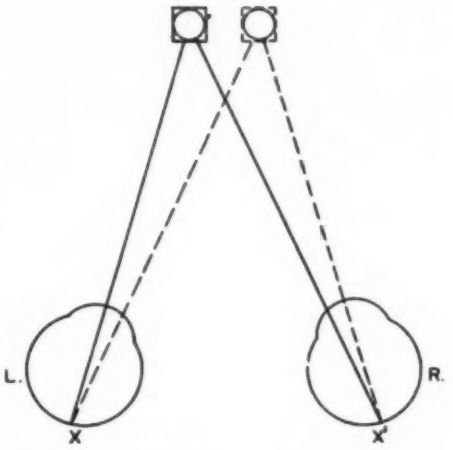

FIG. 4 – Binocular tetralopia in alternating esotropia. Diplopia occurs in each eye simultaneously at the objective angle of deviation.

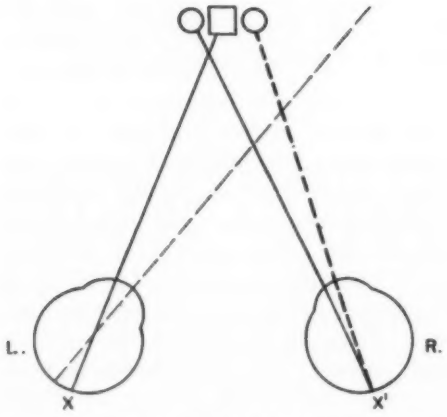

FIG. 3 – Binocular triplopia with alternating esotropia. By requiring the habitually deviating eye to fixate, the image falling on the right fovea (X') is projected straight ahead, and the image falling on the left fovea (X) is projected to the left, producing homonymous diplopia. An awareness of the previous directional projection of the right fovea (X') is still present, and a two-directional projection of the right fovea results in binocular triplopia with alternating esotropia.

Actually, in the beginning of treatment for correction of an anomalous correspondence, we are not working with any part of the deviated eye except the fovea. Only during the diagnostic examination is the off-foveal area in the deviated eye permitted a relationship with the fovea in the fixating eye. In any technique used, a completely new condition, or correspondence, is involved when treatment begins. It is erroneous to assume that treatment is given for the off-foveal area in the deviated eye or that monocular diplopia occurs because the images of the off-foveal area and of the fovea of the nonpreferred fixating eye are seen simultaneously. It is not unusual to find monocular diplopia occurring in the nonpreferred fixating eye when the opportunity for bifoveal fixation is presented at the objective angle, as the old localization of the foveal image is still present at the same time that the new visual direction is becoming established.

It is of interest to note that in alternating esotropia, monocular diplopia has occurred in the nonpreferred fixating eye at the objective angle when anomalous retinal correspondence was not present. We have not found monocular diplopia in unilateral strabismus associated with amblyopia or exotropia. During our twenty years of orthoptic practice, we have not seen a case of primary vertical deviation with anomalous correspondence, and we have seen only one case of exotropia which may possibly have been associated with anomalous correspondence. Frequently a vertical deviation is associated secondarily with an anomalous correspondence. Quite often, in a divergence-excess type of deviation a convergent position may result from an excessive attempt at fusion, but this does not in any way resemble an anomalous retinal correspondence. Upon using the cover-uncover test in these cases, the fixation is steady although the eyes are in a convergent position.

We believe that not only is there such a condition as anomalous retinal correspondence but that there are two types of the condition, which are classified according to foveal development. A classification which we use divides all cases of anomalous retinal correspondence into two groups: group 1 – anomalous retinal correspondence associated with amblyopia; group 2 – anomalous retinal correspondence associated with alternating esotropia. The treatment and response to treatment in these cases varies with each group.

In group 1, the primary treatment consists of an attempt to develop visual acuity of the deviating eye to foveal perception, or as nearly equal acuity in the two eyes as possible. Usually, the only treatments necessary at first are complete occlusion of the fixating eye and amblyopic exercises. These exercises should use planned training material. They can be given at home, with periodic office visits as infrequent as four to six weeks apart *(table I)*. The patient is spared the expense and the time for office visits until binocular instrument work is required. In the majority of cases, until the visual acuity of the deviating eye has reached 20/40, or at least 20/50, suppression is too great for satisfactory binocular training. As foveal visual acuity develops, the former relationship of the eyes becomes less dominant.

With the development of foveal vision accomplished, treatment is not directed to correction of anomalous correspondence, but rather to the stabilization of the binocular function. With bifoveal stimulation on the major amblyoscope and continued complete occlusion of the habitually fixating eye, correction of this retinal correspondence is easily obtained in the majority of these cases; in fact, after the original diagnosis we no longer consider them as cases of anomalous correspondence. In only one out of 58 cases of anomalous correspondence with amblyopia did we find that a set pattern of anomalous correspondence had occurred prior to the onset of amblyopia. In this case, use of the monocular diplopia technique* was necessary to correct the anomalous correspondence after the suppression amblyopia was corrected.

Instability of binocular function found in strabismus is not considered in our classification as anomalous correspondence. It is not unusual to find that a patient with squint, when asked for a subjective finding on the major amblyoscope, will attempt binocular cooperation at an angle of deviation which is less than the objective angle.

*Technique explained under treatment of group 2.

TABLE I

Group 1: Cases of ARC Associated with Amblyopia

AGE AT ONSET, IN YEARS	AGE AT FIRST EXAMINATION, IN YEARS	VISION WITHOUT GLASSES AT FIRST EXAMINATION	OBJECTIVE ANGLE OF DEVIATION (△)	SUBJECTIVE ANGLE OF DEVIATION (△)	TYPE ARC	OFFICE TREATMENTS FOR CORRECTION		VISION WITHOUT GLASSES AT LAST EXAMINATION
						NO.	INTERVAL	
1.5	5.5	R. 20/40–3 L. 20/100–1	25 Et′ 14 Et	10 Et′ 2 Et	Neg.	9	month	R. 20/30+1 L. 20/30
Unknown	10	R. 20/70 L. 20/30+3	16 Et′ 12 Et	7 Et′ 4 Et	Neg.	3	month	R. 20/15 L. 20/15+2
1.25	7	R. 20/100 L. 20/30+2	35 Et′ 28 Et	20 Et′ 16 Et	Neg.	8	month	R. 20/30–3 L. 20/20–2
3	5	R. 20/30–3 L. 20/200	45 Et′ 35 Et	10 Et′ 6 Et	Neg.	10	month	R. 20/20–2 L. 20/30–3
1	6	R. 20/300 L. 20/30+3	50 Et′ 38 Et	25 Et′ 15 Et	Neg.	8	month	R. 20/20–1 L. 20/20–1
2	7	R. 20/30 L. 20/50–1	32 Et′ 25 Et	22 Et′ 18 Et	Neg.	7	month	R. 20/30 L. 20/30–3
4	10	R. 20/40–2 L. 20/30+3	30 Et′ 22 Et	16 Et′ 5 Et	Pos.	22	week	R. 20/30+3 L. 20/20–2
6	8	R. 20/70+2 L. 20/30+3	20 Et′ 12 Et	12 Et′ 4 Et	Neg.	17	week	R. 20/30–2 L. 20/30+3
Birth	7	R. 20/30–3 L. 20/50+	40 Et′ 28 Et	25 Et′ 16 Et	Neg.	10	month	R. 20/20–2 L. 20/30+1
4.5	5	R. 20/50–3 L. 20/30+2	25 Et′ 17 Et	8 Et′ 2 Et	Pos.	21	month	R. 20/30–2 L. 20/30+2

Usually, the subjective angle is not definite in these cases and varies on different attempts. Unless the subjective angle is consistently less than the objective angle, an anomalous correspondence would hardly have had time to become established, especially in the younger patients. In cases of accommodative squint, the subjective angle may be different from the objective angle without the presence of anomalous correspondence.

An observation of the corneal reflections and the use of the cover test and the cover-uncover test while observing the fixation are sufficient to keep the examiner informed of the state of retinal correspondence. When a partial amblyopia is present, there will be found a greater subjective angle of deviation than when the amblyopia is severe. With complete occlusion of the fixating eye, improvement in visual acuity and bifoveal fixation practice at the objective angle of deviation, the anomalous correspondence usually responds to corrective treatment.

During bifoveal fixation practice, it is imperative that bifoveal fixation be maintained. This can best be assured by constantly checking with the cover-uncover test at required intervals. At first, it is surprising how often a slight deviation is present in the nonpreferred fixating eye although the examiner is working at the objective angle. Particularly is this true if a varying vertical deviation is present along with the lateral deviation. It is my opinion that, in some cases, control of the deviation can be definite only with macular and foveal targets. Success in correcting an anomalous retinal correspondence depends to a great extent on the targets used. The choice of targets depends upon several factors: fixation, visual acuity, and the age of the patient. In my experience, best results are obtained by using first-grade foveal-macular targets when visual acuity permits. Complete information cannot be obtained unless one observes conditions when first one eye and then the other fixates the smaller of the two targets used. If foveal-macular targets can be used, the foveal target is placed first on the side of the nonpreferred fixating eye. Foveal targets, 1 to 3 mm. in diameter, and macular targets, 3 to 10 mm. in diameter, or a combination of both, are necessary. For a successful correction, fixation must be confined to the foveas to control an alternating esotropia and a possible alternating sursumduction. Macular targets for the very young patient serve the purpose and are less exacting than foveal ones.

In the beginning of corrective treatment, the field of vision can be confined by fixation of bifoveal targets. Later, larger targets are used to include the paramacular areas and the periphery. When areas of suppression are found in the periphery, they should be eradicated by treatment using a first-grade peripheral target after bifoveal fixation or first-grade single binocular vision occurs spontaneously at the objective angle of deviation. Second-grade targets usually give erroneous findings at first. Frequently, it has been found that when second-grade targets had been used before first-grade binocular vision was obtained, bifoveal fixation was not present, even though the patient reported fusion. By using the cover-uncover test (darkening one eye and watching fixation of the other eye, and then after an interval, determined by steadiness of fixation, repeating this process, darkening the other eye) there will be found a slight movement of fixation, but this movement, no matter how slight, is sufficient to prevent fusion of foveal retinal elements.

The treatment in cases classified as group 2 begins with instrument train-

ing as soon as the diagnosis of anomalous correspondence is made and the patient is old enough to cooperate with the orthoptist. In these cases of alternating esotropia the visual acuity is foveal. Successful corrections have been achieved in patients as young as four years of age and as old as 33; most patients are more than 5 and less than 10. The treatment consists of complete occlusion of the fixating eye and use of the major amblyoscope. Daily treatments are desirable. They require from a half-hour to two hours, depending upon the age of the patient. Correction has been obtained with less frequent visits *(table II)*. In our work for the past sixteen years, the technique utilizing monocular diplopia has been used entirely. By this technique, the preferred fixating eye becomes the deviating eye at a position on the amblyoscope which is outside the subjective angle and at which alternate fixation is spontaneous and homonymous diplopia is elicited. When the habitually deviating eye can maintain foveal fixation at the subjective angle, progress is continued through the angle of anomaly to the objective angle as long as fixation and homonymous diplopia remain. This is followed by bifoveal fixation practice at the objective angle until first and second grades of bifoveal fusion are spontaneous. Peripheral targets are used to eradicate suppression in the periphery.

When fusion at an anomalous subjective angle is demonstrated, the anomalous correspondence is called positive, and when only a crossing of the images is reported, the anomalous correspondence is called negative. This classification is used whether or not monocular diplopia is elicited, as it is the elimination of the anomalous pattern rather than the presence of monocular diplopia which determines the correction. Except for requiring the originally deviated eye to become the fixating eye, the corrective procedure is the same as that for a case of alternating esotropia without anomalous retinal correspondence in which alternate foveal fixation could be maintained on the major amblyoscope from behind the zero position to the objective angle of deviation. By turning the light on the preferred fixating eye off and on, as required, and observing the corneal reflections and steadiness of fixation, the examiner is able to keep foveal fixation under control. After normal retinal correspondence is attained, a certain amount of repetition of the exercises is necessary for the new pattern of association to become an unconscious habit. The length of time for correction depends upon individual conditions.

In cases in which monocular diplopia is elicited in the originally deviated eye at the subjective angle, the patient is instructed to try to maintain foveal fixation with this eye when the cover is removed from the preferred fixating eye and both eyes are uncovered. The patient may not be able to do this at first, and several attempts may be necessary. When suppression occurs upon losing foveal fixation in the deviated eye, it may be necessary to return the target toward zero position until homonymous diplopia and spontaneous alternate foveal fixation are elicited, before making another attempt to maintain foveal fixation with the deviated eye at the subjective angle. This procedure is particularly helpful with children under 8 years of age, and it can be presented in the spirit of a game without frustration to the child. Patients 8 years of age and older when attempting foveal fixation at the subjective angle can be instructed to look for the suppressed foveal image in the nonpreferred fixating eye. This may be found to the right of the nonfoveal image in a nonpreferred fixating left eye

TABLE II

Group 2: Cases of ABC

TABLE II

Group 2: Cases of ARC Associated with Esotropia

AGE AT ONSET, IN YEARS	AGE AT FIRST EXAMINATION, IN YEARS	VISION WITHOUT GLASSES AT FIRST EXAMINATION	OBJECTIVE ANGLE OF DEVIATION (△)	SUBJECTIVE ANGLE OF DEVIATION (△)	VERTICAL DEVIATION	TYPE ARC	OFFICE TREATMENTS FOR CORRECTION*	
							NO.	INTERVAL
3	5.5	R. 20/40 L. 20/40	47 Et' 36 Et	25 Et' 25 Et	Alternating sursumduction	Neg.	12	month
3	6	R. 20/30+1 L. 20/30+1	52 Et' 40 Et	12 Et' 4 Et	3△ right hypertropia	Neg.	22	week
1	10	R. 20/30+2 L. 20/30+3	33 Et' 20 Et	14 Et' 10 Et	Alternating sursumduction	Pos.	14	week
1.5	8	R. 20/40+3 L. 20/40	85 Et' 70 Et	15 Et' 10 Et	4△; 8△ left hypertropia	Neg.	11	8-week
Birth	4	R. 20/50+ L. 20/50+	45 Et' 25 Et	15 Et' 2 Et	3△right hypertropia	Pos.	13	month
0.25	4.5	R. 20/50+2 L. 20/50+3	35 Et' 20 Et	15 Et' 3 Et	2△ left hypertropia	Pos.	7	month
2	8	R. 20/30+ L. 20/30+	46 Et' 36 Et	20 Et' 18 Et	1△ left hypertropia	Neg.	17	week
Birth	4	R. 20/40 L. 20/40	30 Et' 20 Et	16 Et' 5 Et	4△ left hypertropia	Pos.	8	month
Birth	10	R. 20/40 L. 20/40	22 Et' 12 Et	10 Et' 2 Et	2△ right hypertropia	Neg.	12	week
1.5	7	R. 20/30+ L. 20/20–	30 Et' 22 Et	18 Et' 8 Et	1△ right hypertropia	Pos.	8	week
Birth	16	R. 20/20 L. 20/20	65 Et' 50 Et	25 Et' 15 Et	4△ right hypertropia and Alternating sursumduction	Pos.	29	daily
Infancy	20	R. 20/30† L. 20/30†	40 Et' 28 Et	30 Et' 15 Et	Alternating sursumduction	Neg.	7	daily

*All treatments were preoperative except in the third, fifth, and eighth cases, in which no operation was performed, and in the tenth case, in which treatment was postoperative.
†With glasses

and to the left of the nonfoveal image in a nonpreferred fixating right eye.

Although the nonfoveal image and the foveal image in the nonpreferred fixating eye are not seen simultaneously, the patient usually can be made aware of the location of the nonfoveal image. The awareness of these two images is not to be confused with the presence of monocular diplopia, which is a two-directional projection from the fovea resulting in two images being seen simultaneously.

When the patient can maintain a new localization corresponding to the foveal localization in the preferred fixating eye, the examiner's work is less exacting. The patient has learned where to find the foveal image and can make the necessary advancement through the angle of anomaly without requiring as much assistance from the examiner. When monocular diplopia is not present at the subjective angle in the originally deviated eye, alternate foveal fixation is used to reach the objective angle, but the examiner must exercise care to ascertain any movement of the eye inward, especially when the objective angle is reached. In cases in which monocular diplopia occurs at the objective angle instead of at the subjective angle, foveal fixation is required of the nonpreferred fixating eye as well as of the preferred fixating eye.

We might elaborate here on the findings of the examiner at the subjective and objective angles of deviation. In anomalous correspondence, when the cover test is used at the subjective angle, an alternate deviation of the eyes occurs when there is a change in fixation. This is quite obvious. At the objective angle there may be only a slight movement of the suppressed eye when foveal fixation is required. If fixation objects larger than macular targets are used, the movement may be so slight that it is lost to the examiner. Merely reaching an objective angle by cover test does not indicate bifoveal fixation. Bifoveal fixation is exacting, and the patient, not having had this refined experience, needs help for this accomplishment. We must recognize that the possibility of a cure can be determined in this early phase of training. Suppression of the foveas at the objective angle is usually present in all cases of squint, whether the retinal correspondence is abnormal or normal. Eradication of foveal suppression and development of bifoveal fusion are essential for normal use of the eyes. The time required for this development is well spent, as further training without bifoveal fusion limits the patient's chances of a functional cure and invites the recurrence of suppression amblyopia, as well as a possible anomalous correspondence. Frequently, postoperative diplopia is eliminated by bifoveal fusional training.

Since orthoptic training is accomplished only through re-education and the learning process does not cease to function, the age of an individual should not be considered as a barrier to correction of anomalous retinal correspondence. Some of our most satisfying results have been in adults. The time required and his daily duties may deter an adult from taking orthoptic training. However, the time necessary for correcting an anomalous correspondence in a child or in an adult is short in comparison with the time required for single binocular vision to become an unconscious pattern in the normal use of the eyes in daily life. The length of time required to correct an anomalous correspondence depends upon individual conditions, including the foveal development. (Amblyopic adults are not being considered in this discussion.) Whereas an adult requires several hours of treatment daily, in certain instances a young child can receive

half-hour treatments as infrequently as once a month with successful results.

The orthoptic re-education process is sufficiently slow for adjustments to be gradual, so that the adult actually is more comfortable under orthoptic training than when he undergoes the sudden change of surgical correction without supplementary orthoptic treatment. We all know of postoperative cases in which adults are struggling to overcome an annoying diplopia or have an undercorrection that does not give a satisfying cosmetic result. In those adults who have anomalous retinal correspondence and have not received orthoptic treatment, the surgical results are all the more disappointing. The pattern of seeing has been formed so long that, unless re-education of the sensory apparatus has been accomplished prior to surgical intervention, the patient usually returns to the use of his old visual localization and his eye turns in to the preoperative angle of deviation. However, this type of case is rare.

From our files, a thousand cases of squint were studied to determine the status of retinal correspondence.

Patients under four years of age were not included in this study because their tests and treatments on instruments were unsatisfactory. The majority of our patients with esotropia who are under four years of age are treated with occlusion, and we have found that anomalous correspondence was not present in any of these patients when they became old enough for training on instruments. Records prior to the year 1954 were used so that results of treatment could be given. There were 106 cases of anomalous correspondence. These were classified as follows:

1. Anomalous correspondence with amblyopia ..... 58 cases
2. Anomalous correspondence with partial amblyopia ..... 21 cases
3. Anomalous correspondence in alternating esotropia ..... 27 cases

The anomalous correspondence was corrected in 49 cases from group 1; in 17 cases from group 2; and in 25 cases from group 3; that is, in more than four out of five in each category. Possible causes of failure in the noncorrected cases are lack of visual development, discontinuance of treatment by the patient, and removal of the patient from the city. At first these patients made indefinite fusional attempts at various angles of deviation, but these ceased with objective bifoveal fixation practice. All patients achieved *momentary* first-grade foveal binocular vision during the first treatment. It has been most gratifying to find on rechecking the majority of the corrected anomalous correspondence cases from one to twenty years' standing that the anomalous correspondence has not recurred.

SUMMARY

1. In the majority of cases anomalous correspondence can be corrected.

2. A major amblyoscope is required for the techniques given in this discussion. Treatment on instruments usually is not begun until the patient is at least five years of age.

3. Transferring fixation to the habitually deviating eye interrupts an anomalous correspondence. (This is the basis for the monocular diplopia technique.)

4. Bifoveal stimulation by practicing steady fixation and by use of the cover-uncover method at the objective angle is imperative.

5. The objective angle may vary from time to time, and constant rechecking of the objective angle is necessary in cases of alternating esotropia and of alternating sursumduction.

6. First-grade macular and foveal targets provide a direct and necessary control of fixation, especially in cases of alternating esotropia and of alternating sursumduction. Use of second-grade and

peripheral targets follows development of foveal first-grade binocular vision.

7. The cover test and cover-uncover method, along with observation of the corneal reflections, are the examiner's means of determining an anomalous correspondence.

8. A difference between the objective and the subjective angle of deviation does not necessarily indicate the presence of an anomalous correspondence.

9. Complete occlusion of the fixating eye is necessary.

10. Daily treatments are desirable but not imperative.

11. Usually, the younger the patient, the less treatment on instruments is required.

12. In adults when bifoveal visual development is present the prognosis is favorable.

13. In strabismus, anomalous correspondence and monocular diplopia occur only when binocular function is present.

14. Monocular diplopia is a two-directional projection from the fovea (Burian).

15. Monocular diplopia occurs in treatment of anomalous correspondence when the visual direction of the fovea is changed and an awareness of the previous localization is still present.

16. Monocular diplopia does not occur simultaneously from the off-foveal area and fovea of the deviated eye, as has sometimes been erroneously reported.

17. A slight movement of the eye will be observed when the foveal visual direction is changed.

18. When working at the subjective or objective angle, the patient must be taught where to find the second image; that is, to maintain foveal fixation with the nonpreferred eye instead of with the preferred fixating eye.

19. The duration of monocular diplopia varies in accordance with the duration of the anomalous correspondence and the age of the patient. Monocular diplopia can occur at the objective angle with bifoveal fixation, either with or without an anomalous correspondence.

20. In our twenty years of orthoptic practice, we have not had a patient, child or adult, who maintained monocular diplopia upon completion of treatment.

CASE HISTORIES

Case 1. R. W. In this case of alternating esotropia with anomalous correspondence, binocular triplopia was demonstrated at the objective angle; the monocular diplopia occurred when the nonpreferred eye was fixating.

The patient was first examined in July 1953, when he was 8 years old. Onset of squint had occurred when he was 19 months old. Results of the examination were as follows: Visual acuity was R.E., 20/30-3; L.E., 20/30-3. Fixation was alternating, with the right eye preferred. Rotations demonstrated paresis of the lateral recti and the right inferior oblique. Results of cycloplegic refraction were as follows: R.E., +.25 D. sph., +.25 D. cyl., axis 90; L.E., +.37 D. cyl., axis 105. Glasses were not prescribed. Upon orthoptic examination on the major amblyoscope, at the objective angle neither first-grade nor second-grade binocular vision was present. The angle of deviation for near was 86△ esotropia with 9△ left hypertropia; for distance, 80△ esotropia with 7△ left hypertropia. At the subjective angle the images crossed; the angle of deviation for near was 18△ esotropia, and for distance it was 10△ esotropia.

Treatment was begun with complete occlusion of the preferred fixating (right) eye and bimonthly office visits (patient lived out of town). During three months of occlusion therapy, the objective angle and the rotations improved. Alternating foveal fixation was maintained to the objective angle of deviation. Bifoveal fixation practice was given with first-grade foveal and macular targets at the new objective angle: 68△ esotropia with 4△ left hypertropia for near; 60△ esotropia with 3△ left hypertropia for distance. On the seventh visit, monocular diplopia was present at the objective angle in the nonpreferred fixating (left) eye. In this instance, the monocular diplopia occurred while the eye was fixating the smaller of the two macular targets. Simultaneous macular perception was steady and subjectively spontaneous on the eleventh

visit. Second-grade macular and foveal binocular vision developed, followed by training with peripheral first-grade and second-grade targets. Monocular diplopia did not recur after the twenty-third visit. The fusional range was insufficient, and two surgical procedures were necessary. Postoperative orthoptic training was given. The final result of treatment was single binocular vision for near and for distance.

Case 2. C. S. In this case of alternating esotropia, binocular triplopia occurred without an accompanying anomalous retinal correspondence; the monocular diplopia occurred both in the preferred fixating eye and in the nonpreferred fixating eye.

The patient was first examined in September 1949, when he was 6 years old. Onset of squint had occurred when he was 2 years old. Results of the examination were as follows: Visual acuity was R.E., 20/30+1; L.E., 20/-30+2. The fundi appeared normal. Cycloplegic refraction showed R.E., +3.00 D. sph., +.50 D. cyl., axis 90; L.E., +3.00 D. sph., +.50 cyl., axis 90. Glasses were not prescribed, as neither the visual acuity nor the deviation was improved by the refractive correction. The angle of deviation was 85△ esotropia for near and 80△ esotropia for distance, with an alternating sursumduction. Fixation was alternating, with the left eye preferred. Versions demonstrated a lag of the lateral recti and overaction of the inferior obliques. On the major amblyoscope there was simultaneous perception at the objective angle of deviation. The subjective angle of deviation could not be determined. There was no correspondence or association. Surgical correction of the right eye was done in December 1949. This was followed by orthoptic treatment after one month.

Treatment was begun with complete occlusion of the preferred fixating (left) eye. Alternating foveal fixation was maintained to the objective angle. Bifoveal fixation practice was given at the new objective angle of deviation (40△ esotropia for near; 20△ esotropia for distance) on the major amblyoscope with first-grade macular targets. In order to maintain bifoveal fixation, the fixation was checked constantly by turning off first one light and then the other at required intervals. Office visits of 45 to 60 minutes once a day were continued for 28 days. Simultaneous macular perception and monocular diplopia in the right eye occurred simultaneously on the third day of treatment while the left eye was fixating the smaller of the two first-grade macular targets. Faint monocular diplopia occurred in the left eye when the right eye was required to fixate the smaller target. On the fourth day, monocular diplopia in either eye was present when the other eye was fixating a smaller target, but simultaneous monocular diplopia in both eyes (tetralopia) did not occur. The previously variable vertical and lateral deviations became fixed when bifoveal fixation occurred. Complete suppression was present off the major amblyoscope. Monocular diplopia did not recur in the left eye after four more days, nor did it occur in the right eye after seven days. Fusion of second-grade macular targets and a spontaneous homonymous diplopia were present three days later.

Orthoptic training was continued for three years, and habits of normal single binocular vision developed. (Developing bifoveal binocular vision at the angle of deviation on a major amblyoscope is most important, but usually this training is for a shorter period than that used for training off the amblyoscope.) Various forms of occlusion were necessary until sufficient fusional amplitudes and useful single binocular vision developed to prevent the recurrence of foveal suppression.

Case 3. M. E. L. This was a case of alternating esotropia with anomalous correspondence in which binocular triplopia was demonstrated.

The patient was first examined when she was 20 years old, in February 1936. The onset of squint had occurred after the patient had had whooping cough when she was 3 months old. Results of examination were as follows: Visual acuity without correction was R.E., 20/30-3; L.E., 20/30. Cycloplegic refraction showed R.E., +1.00 D. cyl., axis 85; L.E., +1.00 D. sph., +1.25 cyl., axis 105. With this correction the visual acuity in the right eye was 20/20, and in the left, 20/20-2. The angle of deviation was 60△ esotropia with 3△ left hypertropia for near and 50△ esotropia, 3△ left hypertropia for distance. Fixation was alternating, right eye preferred. Versions showed a lag of the lateral recti. The left eye was operated on in February 1936. There had been no preoperative orthoptic examination. Postoperative orthoptic examination was made in May 1936. On the major amblyoscope with glasses worn, neither first-grade nor second-grade binocular vision was present at the objective angle of deviation: 55△ esotropia with 2△ left hypertropia for near, and 45△ esotropia with 2△ left hypertropia for distance. Images fused with anomalous correspondence at the subjective angle of deviation: 5△ esotropia for near and 5△ exotropia for distance. Orthoptic treatment was not given. There was a second operation, this time

on the right eye, in May 1936. Postoperative orthoptic examination was made in July 1936. On the major amblyoscope, with glasses worn, neither first-grade nor second-grade binocular vision was present at the objective angle of deviation: 40△ esotropia with 2△ left hypertropia for near; 30△ esotropia with 2△ left hypertropia for distance. Images were fused, with anomalous retinal correspondence, at the subjective angle of deviation: 2△ exotropia for near; 8△ exotropia for distance.

Our technical reports usually do not include the interesting, but disquieting, psychologic phases of a case. However, this case would not be complete without mentioning the perplexity and concern of the two ophthalmologists and the orthoptist regarding it, to say nothing of the disappointment of the young lady and her family after waiting almost twenty years for her crossed eyes to be straightened! To everyone concerned, the eyes appeared as crossed as they did before the surgical operations. Those of you fortunate enough to have known the skill of my former chief, the late Dr. Grady Clay, and his associate, Dr. Mason Baird, can visualize their reactions. This was their first such undercorrected surgical result, but also their first adult patient with anomalous correspondence, as well as mine. As always, my sponsors' first concern was for the patient, and with this in mind an orthoptic trial was given the patient.

Orthoptic treatment was begun in July 1936, with complete occlusion of the preferred fixating (right) eye. At the objective angle on the major amblyoscope, "macular massage" with first-grade macular targets was given for 30 to 45 minutes. There was no response when larger targets were used. At the patient's request, the time was increased to an hour. Monocular diplopia occurred in the left eye on the seventh day at the time momentary bifoveal perception developed. Objectively, the corneal reflections were normal and "looked" brighter, and the eyes were more expressive in appearance. Bifoveal fixation practice was given every day for three weeks, until subjective first-grade binocular vision was spontaneous at the objective angle of deviation. Foveal fusion was developed and became stable by the end of August. Monocular diplopia was present, but it gradually grew fainter and did not recur after October.

Orthoptic training was continued, and a functional cure was accomplished during the next year. At the last orthoptic examination, in 1954, with glasses worn, and using the Maddox rod, the angle of deviation was 10△ esophoria with 2△ left hyperphoria for near and 7△ esophoria for distance. Vergences were 12△ for near and 8△ for distance on divergence; 115△ for near and 85△ for distance on convergence. Recovery was spontaneous. Single binocular reading and Worth four dots at near and at distance were achieved.

Case 4. A. L. S. In this case of alternating esotropia with anomalous retinal correspondence binocular tetralopia* was elicited at the subjective angles (fig. 4). Monocular diplopia occurred in the nonpreferred eye when fixation of that eye was required at the subjective angle, and upon fixation of the preferred eye at a second subjective angle.

The patient was first examined at the age of 14 years, in July 1945. Squint had been present since birth. Her visual acuity was R.E., 20/20; L.E., 20/20. The fundi appeared normal. Cycloplegic refraction showed R.E., +1.50 D. sph., +.50 D. cyl., axis 15; L.E., +1.50 D. sph., +.50 cyl., axis 180. Glasses were not prescribed. With the prism cover test, the angle of deviation was 70△ esotropia for near and 50△ esotropia for distance. An alternating sursumduction was present. Fixation was alternating, with the right eye preferred. Versions demonstrated a lag of the lateral recti and overaction of the inferior obliques. No preoperative orthoptic examination was made. Surgical correction of the left eye was done in July 1945. Postoperative examination, in November 1945, disclosed an angle of deviation of 50△ esotropia for near and 40△ esotropia for distance. Surgical correction of the right eye was done in January 1946. A postoperative examination was made in June 1946. On the major amblyoscope the objective angle of deviation was 65△ esotropia for near and 15△ esotropia for distance. Images were fused at a subjective angle of deviation of 25△ for near and 15△ for distance.

Treatment was begun with complete occlusion of the right eye and daily visits lasting one to two hours. The monocular-diplopia technique was used. With the left (nonpreferred) eye fixating a faint monocular diplopia was present in the left eye at 24△ esotropia at distance on the second day of treatment. The faint image gradually became brighter, and after eight days, monocular diplopia was maintained through the angle of anomaly to the objective angle. Suppression was strong at the objective angle. Momentary simultaneous macular perception was present on the seventeenth day of treatment. The vertical and lateral deviations varied constantly. Bifoveal fixation practice was given at the objective

*Terminology is that of Burian and Lancaster.

angle. With the right eye fixating, monocular diplopia was present at 15△ esotropia for near on the eighteenth day of treatment. The new image was faint; it gradually grew brighter over several days. Bilateral monocular diplopia (tetralopia) was elicited. Simultaneous macular perception became steadier and held spontaneously on the thirty-fifth day of treatment at the new objective angle of 20△ esotropia with 2△ right hypertropia for near and 16△ esotropia and 2△ right hypertropia for distance. Subjectively, tetralopia was present. Fusion with second-grade macular targets followed. Because of school, the daily visits were replaced with weekly visits. Foveal fusion was not steady until ten months later. Tetralopia was present for twenty-two months, but it did not interfere with the progress of the training. Fusional amplitudes were insufficient, and further surgical intervention was necessary.

Postoperatively, foveal fusion was spontaneous on the major amblyoscope at 2△ esophoria with 2△ right hyperphoria for near and 1△ exophoria with 2△ right hyperphoria for distance. For near, fusion was maintained to −4△ on divergence and to +25△ on convergence. For distance, fusion was maintained to −4△ on divergence and to +15△ on convergence. The patient went off to college before single binocular reading could be developed. However, momentary fusion of printed matter on the bar separator had been achieved, and home exercises were prescribed. College activities, marriage, and motherhood prevented further orthoptic training. In 1953, the patient had binocular vision for distance and a part-time phoria for near. To date, anomalous retinal correspondence and tetralopia have not occurred. In this case the time required for correction was longer than in case 3 because of the marked alternating sursumduction accompanying the alternating esotropia and an unstable condition during the period of adolescence.

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ORTHOPTIC INSTRUCTION COURSES

Nine instruction courses for Orthoptic Technicians, sponsored by the American Orthoptic Council through its Committee on Instruction, will be presented this year by the American Academy of Ophthalmology and Otolaryngology. They will be held on Monday and Tuesday mornings, October 14 and 15, during the Annual Session of the Academy. Tickets will be on sale at the Registration Desk. Price of tickets will be $2.00 per hour.

The courses will be open *only* to (1) members in good standing of the American Association of Orthoptic Technicians and (2) orthoptic technicians who have passed the examinations for certification of the American Orthoptic Council but have not yet been formally elected to membership in the American Association of Orthoptic Technicians. Because of the limited capacity of the available rooms, *it will not be possible to admit physicians to the courses.*

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Editorials

A FELICITOUS COMBINATION

In 1930, the first orthoptic clinic either in Great Britain or in the United States, that of the Royal Westminster Ophthalmic Hospital in London, was opened under the direction of Miss Mary Maddox. In less than a year, it had demonstrated that the need for orthoptics could be met by the aid of technical assistants, and there was already a demand for facilities for training such assistants.

The initial success of this clinic may be attributed to a felicitous combination of personnel and equipment. Miss Maddox had been inspired and trained in the fundamentals of ocular motility by her illustrious father, Dr. Ernest Maddox. In addition, she herself had developed sound techniques with the major amblyoscope, two forms of which had become available commercially in the mid-1920s.

Prior to the development of the major amblyoscope, fusion training had been subjective, carried on mainly by means of the stereoscope or the primitive Worth amblyoscope, with no provision for adequate objective control. Patients' responses, whether reliable or not, had to be depended on and were accepted. In the light of our present-day knowledge and experience, we are

dismayed by the possibility that numbers of individuals may have had an anomalous retinal correspondence reinforced by subjective procedures.

The presence of anomalous retinal correspondence was first evidenced by paradoxical diplopia following surgical operations on the eye. Müller, as far back as 1826, wrote an article on the subject and the phenomenon was subsequently studied by a number of eminent ophthalmologists, notably Tschermak, who devised several instruments and methods for diagnosing it. None of these investigators, however, developed any techniques for treating the condition, nor, so far as I know, were they aware of the attendant hazard of uncontrolled fusion training in its presence.

With the incorporated illumination of the major amblyoscope providing corneal reflections whereby objective and subjective observations could be compared, the danger of unsupervised fusion training was revealed. Miss Maddox was among the first to appreciate that an anomalous retinal correspondence may be reinforced by such training. Furthermore, with the facilities of the major amblyoscope, she developed a technique for combatting this undesirable association and demonstrated that normal retinal correspondence could be established by orthoptic measures. Her observations also led her to classify Worth's first and second grades of binocular vision according to the area of the retina stimulated, as paramacular or peripheral, macular or foveal. Accordingly, she devised an accurate series of major amblyoscope targets for investigating the responses of these various areas.

When, in the formative days, it was suggested that physiotherapists might combine orthoptics with their functions, Miss Maddox maintained that orthoptics should be a specialty in its own right. The present status of orthoptics in Great Britain, the United States, and other parts of the world is justification of her conviction. She was eager for the profession to be established on a recognized basis with specified standards and adequate training centers set up under medical control. Before she gave up active participation in orthoptics for matrimony and family responsibilities, the British Orthoptic Board, supervising the training and certification of orthoptists, was a functioning organization.

In the United States, we lagged behind our British cousins in initiating orthoptics and promoting its valid recognition. When the need for this specialty was appreciated here, we were fortunate to have their example and help in organizing clinics and training centers, and in establishing our own American Orthoptic Council to supervise the training and certification of orthoptic technicians.

Now, in the third decade since the clinic was opened at the Royal Westminster Ophthalmic Hospital, the major amblyoscope, the basic orthoptic instrument in that first clinic, remains the instrument of choice for orthoptic training. It is inconceivable that an orthoptic center could function successfully without one. To Miss Maddox, who pointed the way, our debt remains, and it is incalculable.

ELIZABETH K. STARK

ADULT ORTHOPTICS

When examining orthoptic technicians for certification by the American Orthoptic Council, the writer frequently has been impressed by the lack of familiarity on the part of the candidates

with motility problems found in the adult, especially exophoria associated with presbyopia.

This unfamiliarity probably stems from several causes: (1) Most orthoptists obtain their practical training by working with children. (2) For years orthoptics has been conceived generally as "exercises," especially in the case of children. (3) Because presbyopic patients with muscle disturbances usually are treated in the office of the ophthalmologists, those orthoptists studying in clinics and hospitals do not have the opportunity to observe this group of patients. (4) Most candidates are in the younger age groups, and have not experienced personally the problems encountered by the presbyopic adult.

Exophoria of presbyopia, because of its frequency and its amenability to treatment, is a disease entity which presents a great opportunity for the orthoptist. Without being facetious, one might say that almost every normal individual is a potential patient, since the onset of presbyopia is inevitable. Moreover, because of our increasing population, statisticians predict that within the next twenty years ours will be a nation of elderly people. From a therapeutic standpoint we are fortunate, also, that this type of muscle imbalance will respond most readily to orthoptic measures, and the treatment of these cases can be most gratifying.

"Adult orthoptics" presents a great opportunity which thus far has been largely neglected. For those orthoptists who will manifest an interest in this problem, the rewards can be immense.

WM. E. KREWSON, III, M.D.

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The American Orthoptic Council is composed of three representatives each from the American Ophthalmological Society, the Section of Ophthalmology of the American Medical Association, The American Academy of Ophthalmology and Otolaryngology, and the American College of Surgeons. Four associate members are elected from the American Association of Orthoptic Technicians.

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CLASSIFICATION

1. Accommodation
2. Amblyopia
3. Anomalous retinal correspondence
4. Diplopia
5. Divergent deviations
6. Esotropia
7. Fusion
8. Instruments and devices
9. Miscellaneous
10. Paralysis
11. Physiology
12. Refractive error
13. Stereopsis
14. Surgery
15. Torsion
16. Vergences
17. Vertical deviations
18. Vision

1-1

Alpern, Mathew: *Testing distance effect on phoria measurements at various accommodation levels*, A.M.A. Arch. Ophth., 54:906-914 (Dec.) 1955.

The phoria measurements of eleven observers are recorded by graphs to show the effect of the amount of phoria present when the distance and the accommodation levels are changed.

The apparatus used consists of a haploscope with semisilvered mirrors, lens cells, and lenses of 122 mm. focal length, and a test chart illuminated by a 100-watt bulb, which is viewed only by the right eye of the observer.

With the eyes of the observer in position in the haploscope, a five-diopter vertical prism is placed before the left eye, and he is asked to read the smallest letters visible on the test chart. The arms of the scope having been separately moved so that both points of light are as small as possible, the observer is then asked to align these points vertically so that one is above the other. After this reaction is recorded, the stimulus to accommodation is gradually increased, and the changes occurring are recorded. The testing distance is then varied, and results of each change are recorded.

In all cases the increase in esophoria was in direct relation to the increase of accommodation. However the variance of distance in this test apparently had no direct connection with a change of the phoria; in some cases there was an increase, whereas in others, there was a decrease or no change at all.

This study does not seem to group the phoria changes into any definite categories. The pattern of response seemed to be spread out on a continuum. One important result of these measurements is that the observers who showed some increase in the zero level phoria with a decrease in the test distance could not be distinguished from the other observers with respect to a common factor of age, zero level distance phoria, or refractive error.

The observer who showed the largest increase in the zero level phoria with a decrease in the testing distance was the only one of the observers with strabismus.

At the present time the author feels that results of these experiments cannot be used to provide criteria for diagnosis.

1-2

Christoferson, Kent W. and Ogle, Kenneth N.: *The effect of homatropine on the accommodation-convergence association*, A.M.A. Arch. Ophth., 55:779-791 (June) 1956.

To obtain data for the determination of the quantitative relationship between the change

of stimulus to accommodation and the accommodative convergence, a graph was made so that the motor imbalance caused by forced convergence was imposed upon the changed stimulus to accommodation which caused the same amount of imbalance. The change in equivalent forced convergence revealed that normally 2.4△ of convergence corresponds to 1 D. of stimulus to accommodation. Tests made at intervals after instillation of homatropine revealed an increase in the ratio of the change of accommodative convergence caused by the change in the accommodative stimulus, the (AC)/A ratio; the maximal ratio after one hour was 20/1D., decreasing to normal in 25 hours. The magnitude of the (AC)/A ratio is related to the degree of cycloplegia and the near point of accommodation diminishes in inverse proportion. The studies reveal that (1) there is an increase in the (AC)/A ratio when homatropine is instilled in each eye, and the increase is inversely related to the residual accommodation present; (2) there is a proportional convergence response to a given change in the stimulus to accommodation, as caused by lenses, even though practically no accommodation is possible (there is no increase in accommodative convergence in presbyopic subjects, though normal accommodative innervations do not result in clearing of the retinal image, unlike the increments of convergence when nonpresbyopic subjects are under cycloplegia); (3) the pattern of motor imbalance caused by forced convergence or divergence with prisms tends to be unaffected by cycloplegia. The article infers that perhaps convergence is not reciprocally affected by a stimulus for convergent accommodation through the accommodation-convergence association. The writers state that control of the accommodative reflex probably lies in cortical centers although the initial stimulus must be in the retina, where the amount of innervation may originate as response to blurredness of the images, these responses being subject also to the control of higher cortical processes. The authors' interpretation of the origin and stability of the accommodation-convergence association combines two processes: (1) an innate and unalterable relationship, and (2) a relationship subject to adaptation under environmental stress, similar to a conditioned reflex. Presbyopia, occurring slowly, probably is predominated by the first process. Cycloplegia, being rapid, probably increases activity of the second process.

2-1

Burian, Hermann M.: *Treatment of amblyopia ex anopsia with eccentric fixation*, J. Iowa M. Soc. (August) 1956.

It is the opinion of the author that amblyopia ex anopsia with eccentric fixation does not differ in nature from amblyopia with central fixation. Total occlusion of the better eye is not contraindicated, although one has more difficulty in obtaining cooperation from patients when they are older children. However, patients who have amblyopia with eccentric fixation must be followed more closely than those with central fixation, since their visual acuity, even when improved to 20/20, tends to regress more readily than that of patients who started out with central fixation. When central fixation has been obtained, better results can be expected from temporal occlusion, as well as from surgical straightening of the eyes.

Conclusions were based on the records of 42 patients: 35 with eccentric fixation and, for comparison, 7 with very low visual acuity but with central fixation. Graphs for six cases, showing type of fixation, age of patient, visual acuity at onset, method of occlusion, and length of treatment are presented to document the opinion of the author.

3-1

Hansen, A. K. and Swanljung, Helvi: *Studies on the clinical investigation of retinal correspondence*, A.M.A. Arch. Ophth., 54:744-751 (Nov.) 1955.

This article was prepared on a study of a publication by Ronne and Rindziunski in which they theorize on the pathogenesis of anomalous retinal correspondence and utilize for the first time a test which they call the "prism rack test."

"In this test the eyes of the patient are individually exposed to a vertical bar of light. The patient observes these afterimages, projected on the tangent scale or a wall, and a prism rack with horizontal prisms is moved quickly up and down before one of the eyes." The patient reports that the distance between the afterimages changes while the prism rack is moving. Sometimes the afterimages that were originally normal become anomalous and may be perceived abnormally for some time.

The authors have tried the same procedure, and their findings seem to confirm those of

Ronne and Rindziunski in that there are some patients with strabismus who describe a change in retinal correspondence with the aid of a prism bar. It has not yet been determined whether this finding is of any prognostic importance in regard to the future status of the retinal correspondence.

The phi phenomenon has been mentioned occasionally in the evaluation of anomalous retinal correspondence. It has been believed that this test is so sensitive that in almost all patients with comitant strabismus it could be shown that the phi phenomenon was obliterated. It was not possible for the authors to distinguish between heterophorias and heterotropias by this method. The phi phenomenon was absent only in patients with harmonious anomalous retinal correspondence. This test can be used to differentiate between anomalous retinal correspondence and normal retinal correspondence only when the deviation is corrected with prisms or on the major amblyoscope. The use of this method has little value since this phenomenon is absent only in harmonious anomalous retinal correspondence.

The authors feel that both tests are of value in helping to understand the visual pathophysiology, but are not of value for clinical use for these reasons:

1. The tests require intelligent subjects of at least 7 to 8 years of age.
2. The tests are of no value if the patient tends to suppress.
3. The prism rack test requires special instrumentation.
4. The examinations are very time-consuming and exhausting for both the patient and the examiner.

4-1

Shukla, K. N.: *Acute concomitant squint with diplopia,* Am. J. Ophth., 41:121-124 (Jan.) 1956.

In concomitant squint with diplopia, the diplopia may be due to an insufficiency of both lateral rectus muscles or to a breaking down of an esophoria into an esotropia. The difference between these two types of diplopia is that in cases due to insufficiency of the muscles, the distance between the images increases on looking to the right and to the left, but in cases due to breaking down from phoria to tropia, the distance between the images remains constant in all positions. The position of the images remains constant also in the diplopia of divergence paresis.

A case report is presented and analyzed in which a girl, aged 18 years, developed esotropia with diplopia. Onset was sudden and recovery was dramatic following a few orthoptic treatments.

The author feels that there is a definite group of cases of "acute concomitant squint with diplopia" with characteristics not found in other cases of concomitant squint. The characteristics that distinguish these cases of acute concomitant squint with diplopia from cases of concomitant squint with diplopia but without any evidence of involvement of the lateral rectus muscles are as follows:

1. Sudden onset
2. Well-marked squint
3. Homonymous diplopia in all positions, but easily distinguishable from diplopia of divergence paresis and full-fledged bilateral lateral rectus paralysis
4. Presence of fusion and stereopsis on the synoptophore
5. Evidence of inhibition of both lateral recti
6. Amenability to treatment

4-2

Hill, Howard F.: *Absence of the medial rectus,* Am. J. Ophth., 42:93-96 (July) 1956.

A case is reported in which a 33-year-old woman complained of diplopia of four-years' duration. A nonmalignant tumor, which had extended underneath the medial rectus, had been removed from the inner canthus. The right eye was fully exotropic, with no power of adduction. There was binocular fixation with the head turned to the left.

Surgical intervention was recommended, and the following procedure was subsequently carried out: "The conjunctiva was dissected free of the mass in the inner canthus. This tumor-like mass was cautiously dissected out, and an attempt was made to locate the medial rectus. The former insertion of the tendon was visualized, but careful dissection deep into the orbit failed to locate any portion of the muscle. . . . The lateral rectus was recessed 5 mm. The superior rectus was then exposed and divided with as little trauma to the capsule as possible. The medial half of the tendon was transplanted to the former insertion of the medial rectus. The remaining portion of the superior rectus tendon was moved nasally 4 mm. beyond the nasal border of the superior rectus insertion, and sutured in place. . . . The same procedure was carried out upon the inferior rectus tendon."

The patient obtained useful binocular vision, fair adduction ability of the affected eye, and a good cosmetic result.

5-1

Folk, Eugene R.: *Surgical results in intermittent exotropia,* A.M.A. Arch. Ophth., 55:484-487 (April) 1956.

Surgical procedures were performed on fifty patients with intermittent exotropia for distance, and the results are presented in this paper. The follow-up period varied from 6 months to 6 years. No preoperative orthoptics were given.

Over half of the patients achieved a functional cure without orthoptic training, and it was felt that orthoptics would have helped many others to achieve this result. All the patients had a satisfactory cosmetic result, but six had to have a second surgical procedure.

It was felt that there was no advantage in surgical treatment before the patient reached the age of 5 or 6 years. Many of these patients need postoperative orthoptics, and the patient is usually not ready to cooperate before this age.

"Criteria for classification as a functional result consisted of the following: demonstration of simultaneous macular perception and fusion with some amplitude on the major amblyoscope, and the ability to elicit diplopia both on the major amblyoscope and under room conditions when increasing base-out prism was placed before the right eye for distance and near."

When the deviation is 20 to 25△ or less, a 7 to 8 mm. unilateral recession of the lateral rectus may be the operation of choice. When the deviation is greater than 25△ for distance or near, a bilateral recession of the lateral recti of 7 to 8 mm. seems to be indicated.

"Orthoptics does not seem to be required in every case following surgery, but should be held in readiness. The results of surgical treatment indicate that it is probably the treatment of choice in all cases of intermittent exotropia."

6-1

Nordlow, W.: *Permanent convergent squint — early operation and long-term follow-up,* A.M.A. Arch. Ophth., 55:87-100 (Jan.) 1956.

The author reports on 70 patients with "permanent esotropia" who were operated on within four years of the development of their squint and who had received no orthoptic training, but were given full atropine correction of refractive errors.

He states that after one to three operations between 73 and 77.1 per cent of the patients obtained binocular fixation. Of the 22.9 to 27 per cent who still squinted, "approximately half obtained spontaneous binocular fixation" at a later date. At the conclusion of the period of observation, between 73.5 and 78.1 per cent of the patients possessed binocular fixation; in 55 to 59.4 per cent, functional cure was obtained, with orthophoria or heterophoria compensated by fusion, normal correspondence, amplitude of fusion, stereopsis.

The author states that his investigations revealed that a mechanical factor is the only tangible cause of esotropia in at least 70 per cent of cases.

6-2

Urist, Martin J.: *Esotropia with bilateral depression in adduction,* A.M.A. Arch. Ophth., 55:509-515 (April) 1956.

In this study of 124 cases of esotropia with bilateral depression in adduction, Dr. Urist found by the prism cover test in all positions of gaze that in 68 per cent of the cases the right hypertropia was greatest on looking down and to the right, and the left hypertropia was greatest on looking down and to the left. By the prism cover test, esotropia was greatest in supraversion in 76 per cent of the cases. It was felt that this fact might bear some relationship to the amount of esotropia in the central field of gaze.

When the patient had an esotropia on looking up and an exotropia on looking down, he had a 66 per cent chance of having straight eyes in the central field of gaze. If the patient had an esotropia on looking up and straight eyes on looking down, his chance of having straight eyes in the central field was 44 per cent. When he had an esotropia in both positions, greater on looking up, his chances of having straight eyes in the central position of gaze dropped to 14 per cent.

The lateral rectus muscles have their greatest effect on distance and upward gaze. An insufficiency of these muscles is thought to be part of the reason for esotropia on upward gaze. "The straight eyes and esotropia looking down was postulated as due to an inhibition of the medial rectus muscles in cocontraction with the weak lateral rectus muscles in the attempt to keep the eyes straight in the central field, especially for near."

The hyperopic component was abnormal by large. Most of this group were considered functionally accommodative esotropias for distance as the wearing of glasses had the un-

usual effect of correcting more of the esotropia for distance than for near.

6-3

Jampolsky, Arthur: *Esotropia and convergent fixation disparity*, Am. J. Ophth., 41:825-833 (May) 1956.

Dr. Jampolsky defines fixation disparity as the amount by which visual axes miss exact intersection at the point of regard during single binocular vision, being primarily a type of heterophoria, but occupying intermediate stages between heterophoria and heterotropia. Lack of bifoveal fixation may be due either to a small degree esotropia or a fixation disparity. Both conditions show a small shift on single cover test. The amount of deviation demonstrated on screening does not differentiate between them. Only one type of fixation disparity can be diagnosed by objective tests: when the slightly deviating eye is covered, observation of the eye under cover reveals increased adduction and on removal of cover the eye abducts to its slightly convergent position. Subjective sensory tests do not clearly differentiate between the two conditions as normal retinal correspondence and "fusion" may be demonstrated in both instances; however when abnormal retinal correspondence is found, small degree esotropia is present.

An explanation for the physiologic basis of convergent fixation disparity is given. Due to physiologic nystagmus, exact and steady fixation is not possible in a physical sense. "Correspondence *points* such as the foveas therefore in a physiologic sense are actually small *areas* of correspondence." Fixation disparity is limited by the size of Panum's area at the edge of the most central (foveal or axial) fusionable border. In order to test for convergent fixation disparity as it occurs in the state of nature, targets for haploscopic devices must include foveal as well as peripheral fusionable details, the determination of the amount of fixation disparity being measured by nonfusionable dissimilar details any place in the visual field.

It has been found that patients with a small residual esotropic deviation following the optical correction of a significantly high hypermetropia usually have a convergent fixation disparity. Patients with a postoperative residual small degree convergent deviation usually are found to have a small angle esotropia.

It is of little clinical importance to differentiate these two conditions relative to treatment. "Patients with small degree esotropia cannot be treated, and patients with small degree convergent fixation disparity usually need not be treated." Patients with small angle esotropia remain so permanently, but patients with fixation disparity frequently overcome the anomaly with time and develop stable fusion.

"The importance of studying the differential characteristics of these states rests in increasing knowledge of the differential pathogenesis of convergent and divergent strabismus.

"The study is confined to convergent deviations since similar diagnostic studies do not pertain to divergent ocular deviations."

6-4

Norbis, Atilio L. and Malbran, Enrique: *Concomitant esotropia of late onset*, Brit. J. Ophth., 40:373-380 (June) 1956.

Concomitant squint rarely occurs after the age of 5 or 6. Three clinical types of nonparalytic concomitant esotropia of late onset are classified as follows: (1) late and acute onset in myopes accompanied by spontaneous diplopia and usually occurring as a result of psychical or physical shock, (2) late onset in emmetropes or slight hypermetropes with no apparent cause, (3) late onset as the result of ocular occlusion.

The authors report on four siblings with concomitant convergent squint of late onset. All of these children had good visual acuity, moderate refractive error, normal retinal correspondence, and very poor range of fusion.

The importance of heredity in strabismus is discussed. Anomalies of the position of rest and defective fusion are the factors most likely to be inherited, and it is believed that these factors were the cause for the defects in the cases reported.

6-5

Urist, Martin J.: *Atypical accommodative esotropia*, Am. J. Ophth. 41:955-964 (June) 1956.

The author has presented five case reports and a discussion of certain cases of atypical accommodative esotropia which could be classified as accommodative esotropia for near and other cases which could be classified as accommodative esotropia for distance.

There is a clear-cut separation in the clinical findings of these two conditions. In one

type the near position is grossly affected, with little involvement for distance fixation. In the other type the distance fixation is affected, with little trouble for near.

"The occurrence of such a sharp separation in the involvement for near and distance vision may be clinical evidence in support of the concept of a dual innervational mechanism for control of the eyes – a near mechanism of convergence, near accommodation and miosis, and a distance mechanism of divergence, accommodation for distance and mydriasis."

6-6

Atkinson, George S. and Berry, A. E.: *Management of strabismus in children*, Eye, Ear, Nose & Throat Monthly, 35:577-580 (Sept.) 1956.

In making a diagnosis of strabismus every patient must be carefully studied. The following outline of procedure is used by the authors: (1) history, (2) visual acuity, with and without glasses, (3) gross external examination, (4) muscle balance, (5) fusion, (6) accommodation, (7) fundus examination, (8) cycloplegic refraction. Different methods of these diagnostic tests are discussed.

In treatment, first, a refraction under drops must be done; second, occlusion must be tried; third, training with fusion apparatus must be tried. If no improvement results after one year of nonsurgical treatment, then an operation should be considered. Patients with the alternating type of squint should be operated on as soon as the diagnosis is made.

The purposes of treatment of squint by any method are to develop binocular single vision, to stimulate the vision in an amblyopic eye, and to obtain a good cosmetic result, as well as to help the patient physically and economically.

8-1

Rubin, Milton R.: *A simple fixation light*, Am. J. Ophth., 41:119-120 (Jan.) 1956.

Dr. Rubin points out the advantages of a penlight worn on the examiner's forehead for measuring the angle of deviation in the six cardinal directions of gaze.

The patient's head is stationary and the examiner's head is moved in the six cardinal positions, thus making the axis of the fixation of the patient and the axis of observation of the examiner very close. Even in the two downward cardinal positions, measurements are easier and more accurate because the corneal reflexes and movements can be observed without elevation of the upper lids.

The light can also be used as a handlight when the screen-comitance test is performed.

8-2

Mehta, K. H.: *A new occluder*, Brit. J. Ophth., 40:188 (March) 1956.

A new occluder is presented which is made from soft polyvinyl chloride. It is fleshcolored and has a sucker on the front for attachment to the inside of the glasses, ventilator holes, and a large side-shield which can be cut to fit each patient.

8-3

Schmidt, T.: *Equipment for objective determination of visual acuity according to Goldmann*, Am. J. Ophth., 42:123-126 (July) 1956.

The author describes an objective determination of visual acuity according to Goldmann. "Visual acuity is characterized by the minimum angular distance at which two points are just visible separately (minimum separable). If this perception can be deduced from an involuntary physical reaction of the patient, then we speak of an objective determination of visual acuity. The physical reactions in question are eye movements, optokinetic nystagmus, or optokinetic pendular movements. These last mentioned movements are employed in Goldmann's method of objective determination of visual acuity."

The equipment and the method of examination used are aptly described by the author.

"The method can be employed between visual acuities of 0.1 and 2.0. Possible differences between results of objective and subjective examination vary between 1/10 and 2/10. . . . In general this method cannot be employed for the examination of aphakic eyes."

9-1

Crone, R. A. and Velzeboer, C. M. J.: *Statistics on strabismus in the Amsterdam youth*, A.M.A. Arch. Ophth., 55:455-470 (April) 1956.

The authors conducted an investigation concerning the relative frequency of the different types of squint as defined by the clinical features. Nine hundred and fourteen patients were used in the statistical analysis.

Strabismus was found to be more frequent in girls than in boys.

It was found that the features of squint of early onset showed definite characteristics different from the features of squint of late onset.

It was proved that the high percentage of familial occurrence is caused by the hereditary nature of squint rather than by the high frequency of squint in general.

In cases with a history of birth trauma, the authors found an early onset of the squint and felt that trauma may have precipitated the appearance of "squint caused by hereditary predisposition."

In 13 per cent of the cases a precipitating disease was reported, with measles being the most frequent.

Amblyopia was found in 50 per cent of the untreated cases.

Hypermetropia was considered a "trigger factor" in strabismus. Myopia was of no significance in causing esotropia but was of importance in exotropia. Anisometropia was found in 10 per cent of the cases and was felt to be a precipitating factor.

On orthoptic examination, suppression was found to be most prominent in squint of early onset and in alternating squint. Normal correspondence was often found in squint of late onset and in exotropia. Abnormal retinal correspondence was found frequently and chiefly in small angle squint.

The most remarkable finding of this statistical investigation was felt by the authors to be that all types of and characteristics in convergent squint are hereditary to a practically equal extent, and that in "all types of convergent squint one hereditary, pathological mechanism is at work."

9-2

Parks, M. M.: *Strabismus (review of the literature for 1955)*, A.M.A. Arch. Ophth., 56:138-159 (July) 1956.

A splendid review of the 1955 literature on strabismus has been presented by Dr. Parks in this article.

The author describes the new concepts concerning sensory and motor aspects. In reviewing the sensory complications of strabismus, the treatment of amblyopia, suppression, and abnormal retinal correspondence are discussed from various viewpoints.

Motor complications of strabismus, heterophorias, accommodative esotropia, nonaccommodative esotropia, fixation disparity, divergent strabismus, horizontal noncomitant strabismus, and vertical strabismus are some of the topics presented.

Orthoptists and ophthalmologists will find this article to be an excellent review of the concepts and treatment of strabismus.

10-1

Lebensohn, James E.: *Parinaud's syndrome from obstetric trauma with recovery*, Am. J. Ophth., 40:738-739 (Nov.) 1955.

Dr. Lebensohn presents an interesting case history of Parinaud's syndrome, which had originated at the birth of the patient when delivery had required low forceps and rotation.

On first examination at age 8 weeks the following history and findings were obtained: Since birth the eyes had been constantly in a downward position while awake, though the globes turned up when asleep. Reflex conjugate movements were elicited in all directions but upward. The pupils were dilated, gave no reaction to light, but constricted on convergence. The upper eyelids were extremely retracted. The eyeballs rolled up with passive turning of the head downward. No pathologic condition in media or fundi was seen with the ophthalmoscope. Both parents had compound myopic astigmatism, but no other ophthalmic abnormality.

On periodic examination symptoms proved to be subsiding, and when the patient was two and one-half years old "the pupils were of usual size, and reacted to light; and the eyes appeared and behaved normally in all respects."

The paper is accompanied by excellent illustrations.

A discussion of causes and results in cases of Parinaud's syndrome is presented, including remarks and findings of Duke-Elder, Cogan, Gordon Holmes, and Walsh.

10-2

Schlossmann, Abraham: *Ocular palsies of unknown origin*, Eye, Ear, Nose & Throat Monthly, 34:752 (Nov.) 1955.

There are numerous cases of ocular palsy in which the cause cannot be found even after extensive medical examination. The author points out some of the obscure causes. Many of these palsies improve, with the muscle function returning to normal in from six weeks to three months; therefore the prog-

nosis is usually good. Patching of one eye or use of clip-on prisms may be of help in alleviating symptoms until recovery is complete. Several case reports are given.

10-3

Bedrossian, E. Howard: *Sixth-nerve palsy with past-pointing to the opposite side,* Am. J. Ophth., 40:899-900 (Dec.) 1955.

This paper presents the case history of a 62-year-old white woman who had a right sixth-nerve palsy with primary and secondary deviation. The unusual feature was that with the normal eye fixing she past-pointed and past-walked to the left; however with the normal eye occluded she did not past-point or past-walk at all. She had diplopia only with the eyes straight ahead, and the diplopia increased on looking to the right.

The patient recovered completely in two months after being given a diet for latent diabetes.

Two explanations for past-pointing were discussed, and these would not explain this unusual case.

10-4

Villaseca, Alfredo: *Pseudoparalysis of the medial rectus,* Am. J. Ophth., 41: 124-128 (Jan.) 1956.

Two cases are presented in which a recession-resection operation for convergent squint resulted in divergence and abolition of adduction of the operated eye. By the forced duction test with forceps under general anesthesia, it was shown that the pseudoparalysis of the medial rectus was caused by the resistance of the resected lateral rectus.

As the result of second operations the divergent strabismus and limitation of adduction were corrected in both of these cases. In one case the lateral rectus was retroplaced, and in the other case resection of the medial rectus and retroplacement of the lateral rectus were performed.

10-5

Bedrossian, E. Howard and Lochman, Bernard: *Congenital paralysis of sixth and seventh nerves,* Am. J. Ophth., 41:304-308 (Feb.) 1956.

This paper presents a discussion of the Möbius syndrome. In this syndrome lateral movements of the eyes in adduction and abduction are impossible; there is a bilateral facial paralysis with retention of Bell's phenomenon on attempted closure of the eyes; elevation and depression of the eyes are normal; no vestibular nystagmus can be obtained by the calorie or rotary test; there is retention of limited convergence and pupillary reactions.

Two explanations for the pathologic conditions in congenital cases of this syndrome are discussed: (1) a primary aplasia of the muscle due to a mesodermic defect; (2) a nuclear or ectodermal defect. The forced duction test should be useful in differentiating peripheral muscular defects from a nuclear lesion in the congenital cases.

A case is presented in which it was shown that the pathologic process was peripheral in the muscles.

"A knowledge of the existent peripheral congenital pathologic conditions of the ocular muscles will help prevent misinterpretation of vestibular tests in suspected cases of brain tumor."

10-6

Laughlin, Robert C.: *Congenital fibrosis of the extraocular muscles,* Am. J. Ophth., 41:432-438 (March) 1956.

This paper reports six unusual cases of congenital fibrosis of all the extraocular muscles giving a rare syndrome of complete strabismus fixus which may be called general fibrosis syndrome. "The characteristic features of the syndrome are:

1. Fibrosis of all extraocular muscles.
2. Fibrosis of Tenon's capsules.
3. Adhesions between muscles, Tenon's capsule, and the globe.
4. Inelasticity and fragility of the conjunctiva.
5. No elevation or depression of either eye.
6. Little or no horizontal movement.
7. Both eyes fixed in a position of downward gaze 20 to 30 degrees below the horizontal.
8. Bilateral ptosis.
9. Headtilt backward with chin elevated.
10. The condition is present at birth."

In five of the cases surgical treatment by the author resulted in marked cosmetic improvement.

Surgical procedures in the following order were recommended and discussed: "First operation, elevate one eye. Second operation, elevate second eye and correct ptosis in first eye. Third operation, correct ptosis in the second eye."

10-7

Rucker, C. Wilbur; Dyer, John A.; Smith, Dale C., and Taub, Robert G.: *The causes of acquired paralysis of the ocular muscles,* Am. J. Ophth., 41:951-955 (June) 1956.

A review of 653 cases of ocular palsy was made, and the cases were divided into six groups according to the nerve or combination of nerves in which the paralysis was present. Supranuclear lesions, birth injuries, congenital anomalies, and various disorders of the muscles themselves, such as myasthenia gravis, were discarded. The study was restricted to acquired paralysis of the extraocular muscles caused by lesions of the third, fourth and sixth cranial nerves.

Injury to the head made up the largest group in which the cause for the paralysis could be definitely determined. Over 50 per cent of these injuries were received in automobile accidents. The largest group of cases comprised those of undetermined origin. Explanation for this large group is that when isolated paralysis of a muscle of the eyes is the first manifestation of a disease, determination of the cause may not be possible until other signs appear.

Cerebral neoplasms caused involvement more often of the sixth nerve than of the third or fourth. Occlusive vascular disease accounted for many palsies. Aneurysm most often affected the third nerve, and in most of these cases the pupils were affected. Syphilis caused involvement in either the third or the sixth nerve in only 13 cases. This disease did not cause involvement of the fourth nerve in this series of cases. Multiple sclerosis affected the sixth nerve in 7 cases but did not affect the third or the fourth nerve.

The other causes for the most part were inflammatory.

10-8

Reed, Howard, and Israels, Sydney: *Congenital ocular motor apraxia,* Brit. J. Ophth., 40:444-448 (July) 1956.

One case report of "congenital ocular motor apraxia" is presented, and other similar cases previously reported in the literature are discussed. In this condition all forms of vertical conjugate movements are normal, but lateral conjugate rotations of the eyes show abnormalities. Voluntary lateral gaze toward an object at one side is accomplished by jerking the head toward the object while the eyes rotate in the opposite direction. When the head has turned beyond the object so that fixation is possible, both eyes and the head are straightened to look at it. Involuntary lateral movements are normal. Also associated with this condition are delayed walking, slight defects in coordination, and difficulty in reading.

It would seem that children with this condition suffer from scattered foci of cortical damage. It is possible that congenital ocular motor apraxia may be related to certain types of cerebral palsy. Perhaps during the next few years further studies of cerebral palsy will demonstrate the site of the lesion in this condition.

11-1

Cogan, D. G., and Freese, C. G. Jr.: *Spasm of the near reflex,* A.M.A. Arch. Ophth., 54:752-759 (Nov.) 1955.

Case histories of 16 patients with a spasm of the near reflex are presented and discussed by the authors. Signs and symptoms of this condition are "characterized by intermittent and usually painful convergence, accommodation, and miosis." Patients complained of blurred vision, diplopia, headache, dizziness, pain in the eyes, and difficulty in walking. Many other symptoms present in individual cases are not necessarily related to the eyes. The spasms are usually precipitated by attention directed to the eyes.

In this series of cases the condition occurred most frequently in females; only 3 of the 16 patients were males. Onset was commonest between the ages of 15 and 20 years. The spasm of the near reflex occurred in only two cases in which a neurologic disease was present. It is most commonly attributed to hysteria.

Specific treatment consisted in either atropinization or the use of minus lenses for as long as necessary. Orthoptic training or any procedure which called for continued attention to the patient's eyes was not advised.

11-2

Jampolsky, Arthur: *Characteristics of suppression in strabismus,* A.M.A. Arch. Ophth., 54:682-696 (Nov.) 1955.

The author analyzes characteristics of suppression in strabismus and the clinical measurement of suppression.

Visual suppression is an active internal inhibition and is a type of conditioned reflex. It exists only when the strabismic patient tries to achieve some degree of binocular vision, and it is usually developed to avoid diplopia.

The suppression in strabismus is confined to a specific region or regions, and may be demonstrated primarily for similar contours. It requires a short latent period to become manifest.

The age of the patient is a factor in the development of suppression. After the age of 6 years, the ability to establish new patterns of suppression is greatly diminished.

In order to exact the true status of suppression areas in the patient, it is necessary that the conditions of testing very precisely simulate those of casual everyday seeing. Alteration of any of the variable factors may affect the results.

The author describes his use of the Risley prism test and haploscopic tests and stresses that knowledge of the hemiretinal nature of the border of suppression may be used advantageously in the treatment of strabismus.

11-3

Alfano, Joseph E.: *Spasm of fixation*, Am. J. Ophth., 40:724-730 (Nov.) 1955.

Dr. Alfano defines fixation as the "maintenance of the posture of the eyes by tonic contraction of the ocular muscles so that the retinal images of the object which is fixed fall upon the two foveas."

The mechanism of fixation is broken down into two component parts, a voluntary cortical mechanism for coarse adjustment and an involuntary reflex subcortical mechanism for finer adjustment. The pathways of these mechanisms are defined and illustrated by diagram.

When the voluntary fixation mechanism is abolished or destroyed, the reflex mechanism is exaggerated or intensified, resulting in a spasm of fixation. The spasm is started and prolonged by proprioceptic impulses from the ocular muscles, resulting from continually acting retinal impulses upon the uncontrolled cortical mechanism.

Spasm of fixation is characterized by the following conditions:

(1) Paresis or paralysis of voluntary conjugate lateral or vertical movements and voluntary convergence

The patient is unable to look to either side on command, or to an object which he can see placed on either side of him. He turns head in direction in which asked to look. At times he is able to look to one or both sides by moving his eyes in a series of irregular jerky movements, requiring much effort and accompanied by distress. Although he is unable to turn his eyes on command or at will, his eyes occasionally move involuntarily in all directions. There may be an associated paralysis of convergence, but usually normal convergence occurs if the object is brought near slowly.

(2) Intensification of fixation mechanism

Although voluntary movements are impossible, the patient can follow an object which he has already fixed if the object is moved slowly and uniformly. However, in order to move the eyes to follow the object, he must first interrupt the fixation reflex by blinking, turning and jerking his head.

(3) Retention of the proprioceptive reflexes

Although voluntary movement of his eyes from side to side or up and down are impossible, full lateral and vertical excursions of the eyes may be obtained by passively rotating the head.

A detailed and illustrated case history is given. The author summarizes it as follows: "A case of spasm of fixation is reported in which the cause was thought to be damage to the frontal oculogyric centers resulting from hemorrhage from the superior sagittal venous sinus incident to birth trauma."

11-4

Verhoeff, F. H.: *The so-called blindspot mechanism*, Am. J. Ophth., 40:802-808 (Dec.) 1955.

The author critically reviews the Gifford Lecture delivered by Dr. K. C. Swan in 1954 on "The Blindspot Mechanism in Strabismus."

Dr. Verhoeff feels that in many instances incorrect and ambiguous wording occurred. The words "utilize" and "mechanism" are said to be incorrectly used because they imply purposeful actions. To "utilize the blindspot mechanism" would falsely imply that the amount of deviation is changed so that the image would fall upon the blindspot of the deviating eye.

The actual existence of the so-called blindspot mechanism cannot be proved simply by demonstrating the amount of esotropia. Placing a prism of considerable strength over the deviating eye does not necessarily produce diplopia. Swan assumed that if diplopia was the result, the image had been on the optic disc of this eye.

According to Swan, patients with good vision and normal correspondence in whom diplopia can be alleviated only by the use of the blindspot machanism are said to have the

blindspot syndrome. Such patients, he said, are relatively free of symptoms but may occasionally experience diplopia or confusion of images. Verhoeff proved by a simple experiment that no one could be considered asymptomatic under such conditions. In any type or degree of squint if the patient has normal correspondence and is free of diplopia, widespread suppression is present. Since the optic disc is so far from the fovea and is represented by such a relatively small area in the visual field, the blindspot could play only a small part in preventing diplopia. In the opinion of the author, the so-called blindspot mechanism and so-called blindspot syndrome do not really exist, and if they do exist, they are not significant as far as treatment is concerned.

11-5

Tani, George T.; Ogle, Kenneth N.; Weaver, Rex W.; Martens, Theodore G.: *On the precise objective determination of eye movements,* A.M.A. Arch. Ophth., 55:174-185 (Feb.) 1956.

Using a haploscopic device combined with an ophthalmograph camera, which recorded simultaneously the corneal reflexes and the actual positions of the haploscopic targets, the authors tested twenty subjects. The purpose of the experiment was to try to obtain a continuous record of the convergence of the eyes with the actual convergence of the test targets. A discrepancy was found between the amount of convergence of the eyes and that of the test targets. The authors showed that the reason for this discrepancy was that the eyes do not move about a fixed axis, but move along curves associated with translatory displacements. Since the discrepancy was greater after the initial 10 diopters of convergence, they felt that the discrepancy might be due to an exophthalmos and nasal translation of the globe caused by contraction of the inferior and superior oblique muscles which, with increased convergence, enter their field of greatest action. Forced convergence or divergence and accommodative reflexes modified the tonic states of the extraocular muscles and introduced a variability which made precise measurements impossible.

It was proved, however, that precise measurements could have been obtained if the eyes had turned about a fixed axis. This part of the experiment was done by placing steel balls which turned about the fixed axis in the position of the eyes and recording their movements.

11-6

Magee, Alfred J.: *The electromyogram of the lateral rectus muscle,* Am. J. Ophth., 41:275-285 (Feb.) 1956.

The method used by the authors in obtaining electrograms of the lateral rectus muscle of the human being is described. The motor unit is the physiologic unit of contraction and is regulated by the amount of motor-unit firing at a given time. The evidence presented in this paper "would indicate that the motor unit of human eye muscle tissue is about 15 microvolts to several times this in amplitude and about a millisecond in duration."

Electrograms were made of the lateral rectus while the subject gazed at infinity, while he maintained fixation straight ahead, and during abduction, depression, elevation, convergence and divergence.

While the subject gazed at infinity, motor unit firing was at a minimum. During fixation straight ahead there was a definite amount of firing. In abduction, the motor unit firing was proportional to the amount of abduction. In depression and elevation the motor unit firing was similar to that with the eyes straight. In convergence and divergence the electrograms showed oscillating movements of the globe with alternate contraction and relaxation of the muscle until the point of stabilization is reached.

11-7

Hallden, Ulf: *An optical explanation of Hering-Hillebrand's horopter deviation,* A.M.A. Arch. Ophth., 55:830-835 (June) 1956.

The author demonstrates that the Hering-Hillebrand's horopter deviation might be caused by dioptric asymmetries of the eyes. His research is based on the definition of the horopter "as the locus of the points in space whose retinal images fall on corresponding points of the two retinae." By experimental methods the shape of the longitudinal horopter has been determined, and the curve found in this way is called the empirical horopter. According to Hering the empirical horopter does not coincide with the Vieth-Mueller circle; these findings were confirmed by research done in later years. The Hering-Hillebrand deviation is the difference between the empirical horopter and the Vieth-Mueller circle. Hillebrand gave the current explanation of the Hering-Hillebrand deviation. His theory explains not only the horopter deviation but also

a monocular symmetry, the well-known bisectioning experiment of Knudt.

11-8

Harwich, Harry, and Linfesty, John W.: *Pseudostrabismus caused by heterotopia of the macula,* A.M.A. Arch. Ophth., 56:194-206 (Aug.) 1956.

A rare condition known as idiopathic heterotopia of the macula is described in which there is an actual physical derangement of the relationship of the blindspot, or optic papilla, to the macula. A review of the literature on this condition revealed eleven cases which are discussed briefly. In addition to the heterotopia, in many of the cases were found peripheral pigmentation of the retina, anomalous courses of the retinal vessels, persistence of the hyaloid artery or its remnants manifested as membranes or floaters, and a peripheral colobomoid lesion. The relatively high incidence of true strabismus with this condition would suggest that there is a fault in development which might predispose to both the strabismus and the heterotopia.

The authors present one case report of a 25-year-old man with a comitant right hypertropia and exotropia. Fundus examination showed the disc to be elongated below "by a sheaf of white fibers springing inferiorly and partially obscuring the vessels ...In addition to the sheaf effect, one gets the impression from the coloring of the disc and the position of the vessels that the right disc has rotated half a turn clockwise, so that its nasal portion may now be inferior. The major division of the veins shows sheathing. On looking lateral to the disc, no macular area is seen, but merely a normal retinal pattern. Further examination shows that the macula is inferior and medial to its normal position, being about two disc diameters from the disc in the 7 to 8 o'clock meridian. This macula appears normal in every respect except its position. On continuing outward in this meridian, some pigmentation and depigmentation is seen, as in an old localized chorioretinitis. Then, more peripherally and almost straight inferior from the disc, a white tiered, lobulated lesion is seen."

In the opinion of an embryologist who was consulted on this case, the changes causing the apparent rotation of the retina could have been either developmental or mechanical. Congenital displacement of the macula could result from abnormal changes in the relative rate of growth in different parts of the retina. Mechanical displacement of the macula could result from traction such as from adhesion of blood vessels in the primary vitreous to the inner surface of the retina or by the proliferation of retinal strands such as would follow fetal retinitis.

11-9

Keiner, Gerard B. J.: *Physiology and pathology of the optomotor reflexes,* Am. J. Ophth., 42:233-251 (Aug.) 1956.

This paper presents discussions of three aspects of the optomotor reflexes: development of the optomotor reflexes, disturbances in the development of the monocular optomotor reflexes, and disturbances in the development of the conjugated optomotor reflexes.

In considering the development of the optomotor reflexes the author mentions two forms in which the reflex eye movements evoked by nonoptical stimuli are manifested: one is the monocular reflex evoked by proprioceptive impulses emanating from the ocular muscles of each eye separately, and the other comprises the conjugate, compensatory reflexes which are evoked by vestibular and musculosensory stimuli from the labyrinth and the neck muscles respectively. The normal development of these two types of reflexes is discussed in detail.

A disturbance can occur in the development of the monocular reflexes or of the conjugate reflexes, and both will be disturbed in their expressions, although perhaps to different degrees.

The reflex theory regards strabismus as being the consequence of a disturbance in the physiologic building up of the optical reflex apparatus which has the task of regulating and ensuring the position and function of the two eyes under all conditions. It has been shown that this disturbance must lie in the monocular optomotor reflexes. It is also thought that the syndrome of alternating hyperphoria must be due to a defective development of the monocular optomotor stimuli from the lower nasal quadrant of the retina. The above two theories are discussed in detail.

Pendular nystagmus and latent nystagmus are regarded as due to disturbances in the conjugate optomotor reflexes. The author's thoughts on the relationships of these two types of nystagmus to the development of the conjugate optomotor reflexes are presented.

11-10

Alpern, Mathew, and Ellen, Paul.: *A quantitative analysis of the horizontal movements of the eyes in the experi-*

ment of Johannes Mueller: I. Methods and results, Am. J. Ophth., 42:289-296 (Oct.) 1956.

Johannes Mueller was the first to provide evidence that the functions of accommodation and convergence are related. He found that when one eye was occluded and the other eye changed fixation from a far to a near object along the same line of sight, the occluded eye moved nasalward.

The authors have conducted experiments along this line in an effort to gain a better understanding of the relationship of the functions of accommodation and convergence.

Ocular rotational potential was used to study the change in the position of the eyeball during Mueller's experiment. A detailed description of the author's method of obtaining and recording the changes noted in far to near fixation is given.

The results suggested that the far-to-near change in focus occurs as a smooth continuous inward rotation of the occluded eye. Only the occluded eye moves. The evidence in this paper suggests that "the eye movements in the experiment of Johannes Mueller (monocular fixation) follow the 'principle of least muscular effort.' In binocular far-to-near focus, however, or in prism vergence testing the eyes move according to Hering's law of equal innervation of the two eyes." These results imply that if Hering's law remains valid, then the way in which the yoked movements are damped in binocular asymmetrical convergence is essentially different from the way the yoked movements of the fixating eye are damped in Mueller's experiment. Possible explanation of this difference is discussed.

11-11

Alpern, Mathew and Ellen, Paul.: *A quantitative analysis of the horizontal movements of the eyes in the experiment of Johannes Mueller: II. Effect of variation in target separation,* Am. J. Ophth., 42:296-303 (Oct.) 1956.

This paper is a continuation of the immediately preceding one, and its purpose is to give a description of the way in which the various characteristics of the horizontal movement change with a change in the separation of the far and near targets.

Differences between the far-to-near and the near-to-far shifts occur. The latter is a smaller, slower movement, and at the smaller target separations, a slightly lower average acceleration. The amplitude, average velocity, and average acceleration for both types of movements generally show the same characteristic changes as the target separation increases:

(a) Increase in target separation causes increase in amplitudes of these movements, except at the larger target separations where the amplitude appears to reach a maximum and no further increase occurs.

(b) Increase in target separation is associated with an increase in the average velocity of the movement. At six diopters' difference in the stimulus to accommodation the increase of the average velocity reaches a maximum value, and beyond this point no further increase in average velocity of movements occurs in spite of further increased target separation.

(c) Increase in target separations was associated with an increase in the average (negative) acceleration of the target up to a difference in the stimulus to accommodation of six diopters. Further increase in target separation was not associated with further increase in average acceleration of the movements.

"There is no very obvious change in the reaction time with increase in target separation. The differences between reaction times in the far-to-near shift and the near-to-far shift seem to show no constant trend."

Theoretic implications of the results in this paper are discussed.

14-1

Cooper, Edmond L.: *Muscle surgery and orthoptics in the treatment of comitant nonaccommodative strabismus,* Am. J. Ophth., 40:883-890 (Dec.) 1955.

The author evaluated 382 cases of comitant esotropia and 101 cases of comitant exotropia, in all of which surgical procedures had been done. In the cases of comitant esotropia, the highest percentage of successful results was obtained in children under the age of six. After the child is six years old, functional cure is less likely and usually is obtained only with the aid of orthoptics. In comitant divergent strabismus, functional cure can generally be obtained in patients of any age.

The author favors early symmetric surgical procedures in all cases in which he feels that there is a good chance for a functional cure. Nonsymmetric procedures are employed when only a cosmetic cure is expected.

The presence of anomalous retinal correspondence, anisometropia, and a high vertical component lessen the chances for a functional cure. Of the 382 cases of comitant convergent

strabismus, there were five with more than moderate anisometropia, 74 with a definite vertical anomaly, and 93 with an anomalous retinal correspondence. In these cases the results after operation were not encouraging.

When vision is equal and the patient is alternating freely, the percentage of successful results after operation is considerably higher than when the operation is performed when vision is not equal and alternation is not present.

Cooperation between ophthalmologist and orthoptist is stressed as being a large factor in obtaining a successful functional cure.

14-2

Whitwell, John and Preston, Audrey: *Use of miotics in squint surgery,* Brit. J. Ophth., 40:96-99 (Feb.) 1956.

In a number of cases of convergent squint with a residual squint for near but eyes straight for distance postoperatively, pilocarpine was used as an aid in the orthoptic training.

One per cent pilocarpine drops were instilled in both eyes three times a day, and the treatment was started on the second or third day after operation. This treatment was usually continued for four weeks, then the administration of drops was reduced to twice daily for two weeks, then to once daily for two weeks. This routine was varied according to the progress of the patient.

Two factors necessary for successful results are good binocular vision with fusion and fairly good stereopsis. When these factors are lacking there is only a temporary reduction in the angle of squint while the drops are being used.

14-3

Urist, Martin: *The surgical treatment of esotropia with bilateral depression in adduction,* A.M.A. Arch. Ophth., 55:643-665 (May) 1956.

In this paper Dr. Urist discusses the results of surgical intervention in 65 cases of esotropia with bilateral depression in adduction. In this group of cases the esotropia is greater for distance than for near and is greater in upward gaze than in downward gaze. The NPC is good to fair. There is a right hypertropia on looking to the right and a left hypertropia on looking to the left.

The author presents the following six different surgical procedures, and discusses the techniques and results of each:

1. Resection of one lateral rectus.
2. Bilateral resection of the lateral recti.
3. Bilateral recession of the medial recti.
4. Recession of medial rectus and resection of lateral rectus of one eye.
5. Recession of both medial recti and resection of one lateral rectus.
6. Resection of both lateral recti and recession of one medial rectus.

The average reduction in amount of esotropia on upward gaze was greater after a resection of one or both lateral recti than the average reduction in the amount of esotropia on downward gaze. Recession of one or both medial recti produced a relatively greater effect on downward gaze. The amount of correction obtained following a recession of the medial rectus is greater than that following a corresponding resection of the lateral rectus, even for the amount of esotropia present on upward gaze.

Following surgery in 40 of the 65 cases the eyes were straight in primary position, in 16 cases there was undercorrection, and in 9 there was overcorrection. In only two cases did the eyes become straight in all positions of gaze. In 25 cases the eyes were straight in primary position but there was an esotropia in upward gaze which balanced an exotropia in downward gaze. This was seen not only postsurgically but also in many accommodative cases. "A psycho-optical reflex has been postulated for this balance in the straight-ahead position of the eyes."

14-4

Swan, Kenneth C.: *Resection under Tenon's capsule,* A.M.A. Arch. Ophth., 55:836-840 (June) 1956.

In the usual procedures for resection of a horizontally acting muscle, both Tenon's capsule and the conjunctiva are incised parallel to the muscle insertion. The flap of Tenon's capsule over the muscle generally is allowed to retract with the recessed muscle. This flap often forms a mass under the conjunctiva, reducing the effectiveness of the procedure. A newer method is to resect the muscle in order that the normal relationship of Tenon's capsule to the conjunctiva may be retained. The conjunctiva is incised and stripped back to expose Tenon's capsule, which is then incised parallel to the muscle and retracted. The reflections of the muscle capsule to Tenon's capsule are severed. The intermuscular membrane is buttonholed above and below the muscle, and a hook is passed beneath the muscle. O'Brien lock stitches are placed in the mus-

cle before it is resected. Sutures are passed through the edge of the scleral stump to approximate accurately the cut end of the muscle with the old insertion; the knots are tied on the scleral side to minimize pressure on muscle fibers. Tenon's capsule is closed with fine absorbable sutures, the knots being placed beneath the capsule. The conjunctiva is then sutured.

14-5

Fink, Walter H.: *Pitfalls in strabismus surgery,* A.M.A. Arch. Ophth., 56: 265-273 (Aug.) 1956.

This paper is a discussion of the causes of unsatisfactory results in the surgical treatment of strabismus cases. It is divided into three parts: (a) general considerations; (b) factors applying to all the muscles; (c) factors applying to the individual muscles. Detailed discussion and suggestions are given regarding each of these three phases of surgical treatment.

14-6

Sugar, Saul: *The surgical treatment of divergence-excess types of intermittent exotropia,* Am. J. Ophth., 42:619-620 (Oct.) 1956.

Three case reports of intermittent exotropia are given in which the deviation was markedly greater for distance than for near. Surgical treatment in each of these cases included recession of both the lateral and medial rectus muscles. The results from this surgical procedure have been very encouraging.

15-1

Pascal, Joseph I.: *Effect of version and vergence movements on ocular torsion,* Am. J. Ophth., 40:735-737 (Nov.) 1955.

Restating the well-established facts that versions refer to the movements of both eyes in the same direction and that vergences refer to the movements of the two eyes in opposite directions as in convergence and divergence, the author presented an analytical study of the associated torsional movements.

In versions, torsional movements are symmetrical and are seen only in the oblique directions of gaze, with levotorsion on looking up and left, and dextrotorsion on looking down and left. Version movements from primary position to straight up, down, right or left are free of torsion.

On convergence we find asymmetrical torsional movements of both eyes. When the eyes are converged from primary positions at six meters there is some extorsion of each eye, that is, there is dextrotorsion of the right eye and levotorsion of the left eye, with the extorsion increasing as convergence increases. However, extorsion decreases as the plane of regard is lowered, until there is slight intorsion of each eye. When the eyes are raised in convergence, extorsion increases.

In summarizing his study, Dr Pascal stated that torsion of the eyes takes place on version movements in oblique directions and on convergence; but they are different kinds of tortion. The secondary action of the medial rectus was suggested as the mechanism causing the reversal of torsion.

16-1

Westheimer, Gerald and Mitchell, Arnold M.: *Eye movement responses to convergence stimuli,* A.M.A. Arch. Ophth., 55:848-856 (June) 1956.

The experiments described in this article were performed for the purpose of ascertaining the time characteristics of the eye movements elicited by the presentation of visual stimuli in horizontal disparity. The authors refer to the known convergence and divergence movements to re-establish fusion when at a fixed level a stimulus is presented in crossed or uncrossed disparity. Movements of convergence and convergence relaxation were investigated. The term convergence relaxation is used to denote eye movements observed while the two lines of sight change from a converged position to one of lesser convergence, but not of complete divergence. It is so used in order to reserve the term divergence for denoting the movement bringing the lines of sight into a diverged position.

The experiments were performed on three normal young adults. Methods and results are described, together with explanatory charts and drawings of the material used. The experiments were planned to investigate three aspects of the problem:

1. Does the subject carry out a movement of convergence or of convergence relaxation when the only stimulus given to him is disparity?
2. How much time is required for convergence movements and convergence relaxation movements?
3. How much asymmetric convergence is

performed when a far and a near object are both in the line of sight of one eye?

Attention was given to all these questions. An interesting finding is that there was never more than a fleeting moment of diplopia following the stimulus change, in spite of the fact that it required almost a second before the convergence approached the required level.

17-1

Ruedemann, Albert D., Jr.: *Scoliosis and vertical muscle imbalance*, A.M.A. Arch. Ophth., 56:389-414 (Sept.) 1956.

The author presents twelve case histories of patients with a vertical ocular muscle imbalance which produced spinal scoliosis.

Head tilting may be induced by involvement of any one or of several of the vertically acting muscles, and this in turn causes a compensating shift of the spine to neutralize the cervical angulation.

In each of the author's cases the muscle imbalance had been present at the patient's birth. In every case studied the measurements of the right and left legs were equal. No bony abnormality of the vertebral column or the pelvis was found in any case, therefore the scoliosis was muscular in origin.

The cases are divided into four groups:

Group I. Superior oblique-superior rectus syndrome (superior oblique primary muscle at fault).

A. Those maintaining fusion in the primary position, with hyperphoria for near greater than for far, result of the Bielschowsky head-tilting test positive, to the paretic side, underaction of the superior rectus noted in its cardinal position.

B. Those unable to maintain fusion in any position of gaze, with vision equal, horizontal deviation present as well as a hypertropia of the same type in all fields of gaze.

Group II. Superior rectus-superior oblique syndrome (primary superior rectus underaction, superior oblique underaction of ipsilateral eye). These cases are similar to those in group I except that the hypertropia is greater for distance than for near, and the result of the Bielschowsky test is negative.

Group III. Inferior rectus-inferior oblique syndrome (primary inferior rectus underaction with contralateral inferior oblique underaction). Fusion may or may not be present. The head tilt is to the same side as the oblique weakness, and the result of the Bielschowsky test is negative.

Group IV. Inferior oblique-inferior rectus syndrome. (Opposite of Group III). Fusion may or may not be present in the primary position.

The majority of cases are due to superior oblique weakness rather than to superior rectus weakness; however, the two muscles are involved together in many cases, presenting a superior oblique-superior rectus syndrome. Confusion between a superior rectus involvement and a superior oblique involvement may occur when the patient alternates or is fixing with the paretic eye, and also when the patient can fuse in the primary position. "In any case, inhibitional palsy of the contralateral antagonist—superior rectus—may occur." Emphasis is placed upon performing the Bielschowsky head-tilting test correctly, and the procedure is described. Esotropia is present in many of these cases possibly due to excessive adduction with loss of superior oblique abducting power.

"The ocular abnormality in every case was first noted shortly after birth. . . . Therefore the desire for fusion causes adjustment of the entire body above the pelvis. This adjustment is made by the muscles of the back and neck as an attempt to maintain the vertical body position which is required for normal locomotion and balance. Even though fusion is not maintained and there is no bony abnormality, the scoliosis does not disappear. This may be due to the continued demand for cyclorotatory parallelism."

Roentgenograms made with the patient standing and lying showed the scoliosis to be more pronounced in the standing position.

There are several types of vertical muscle imbalance in which scoliosis usually is not noted. These types fall into the bilateral hypertropia or double hypertropia groups.

Although treatment of these cases is not a part of this paper, it is stated that treatment must be directed primarily at the cyclorotatory defect.

17-2

Foulds, Wallace S.: *Post-inflammatory ectopia of the macula resulting in apparent hypertropia*, Brit. J. Ophth., 40:571-573 (Sept.) 1956.

A brief review of possible etiological factors in ectopia of the macula is given. Among the causes are an alteration in the rate of growth of the posterior pole of the eye during the early postnatal period, developmental and congenital anomalies associated with other eye defects, and traction on the retina by post-inflammatory fibrous bands which may result

from either a prenatal or postnatal inflammatory lesion.

A case report is presented in which there was a vertical displacement of the macula by a postinflammatory fibrous band. Full binocular vision was retained although there was the appearance of hypertropia.

18-1

McNeil, N. L.: *Patterns of visual defects in children*, Brit. J. Ophth., 34: 688-701 (Nov.) 1955.

A study was made of the visual defects found in the children in a town with a population of 75,000. The majority of children of school age were referred to the clinic as a result of a medical inspection which included a visual test. The cases were analyzed according to sex, age, and age at first visit to the clinic.

Squints and their characteristics are described. The refractive errors in the nonsquinting cases are classified, and the cases in which there were varying degrees of amblyopia are tabulated.

18-2

Weale, R. A.: *Problems of peripheral vision*, Brit. J. Ophth., 40:392-415 (July) 1956.

Various aspects of the function of retinal periphery have been considered. Diffraction, sensitivity, color vision, and electrophysiology are discussed in detail, and it is concluded that peripheral vision is a difficult subject to study. "It would appear that some consideration of the properties of the retinal periphery may lead to increased visual hygiene, if not also to greater visual efficiency."

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